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BRONCHIAL INVOLVEMENT IN METASTATIC PULMONARY MALIGNANCY

DONALD S. KING, M.D., AND BENJAMIN CASTLEMAN, M.D.
BOSTON, MASS.

THE belief that neoplastic metastases from other parts of the body to the lungs do not involve the bronchi is not always proved by fact. In the past we have been prone to assume that a patient with pulmonary malignancy who raised blood or bloodstreaked sputum or who had evidence of bronchial occlusion was suffering from a primary neoplasm, but there have been enough proved cases of hemoptysis and bronchial obstruction from metastatic growth to make it worth while investigating the actual incidence of bronchial invasion by pulmonary metastasis.

There are two main methods by which a bronchus can be affected by metastatic tumor. First, there may be direct extension from a parenchymal or mediastinal tumor nodule into the wall of a bronchus with eventual mucosal ulceration (Fig. 1) and second, there may be direct metastasis to the wall of the bronchus with the formation of a mucosal metastasis alone (Fig. 2). From either of these, the mucosal involvement may grow large enough to obstruct the lumen of the bronchus and even project down the bronchial tree as an intraluminal plug.

That hemoptysis may be associated with a metastatic pulmonary tumor, but treated as a primary tumor, is illustrated by the following case.

From the Departments of Medicine and Pathology, Massachusetts, General Hospital.
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A man 63 years of age was admitted to the Massachusetts General Hospital in July, 1939, with complaints of (1) pain in the upper right arm of three months' duration, (2) raising of bloodstreaked sputum of two months' duration, and (3) severe pain in the left chest near the sternum of one month's duration. The diagnosis of carcinoma of the

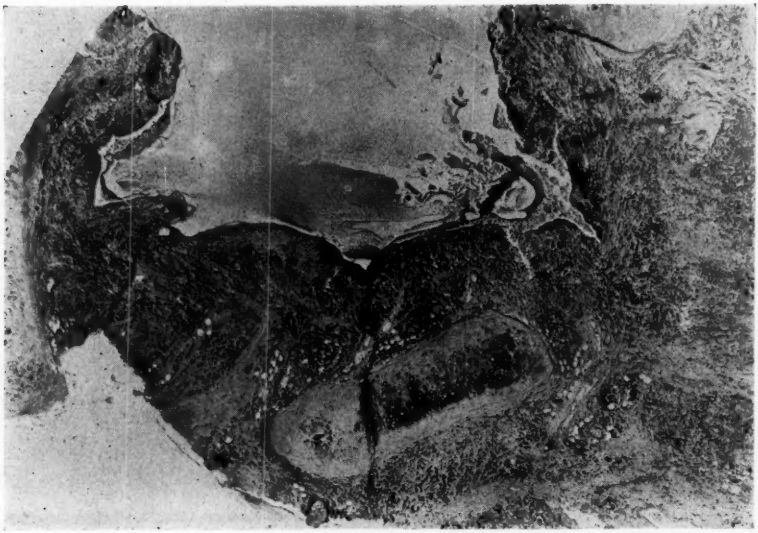

Fig. 1.—Photomicrograph of bronchus, the wall of which is infiltrated with metastatic cancer. In a few places the mucosa is ulcerated.

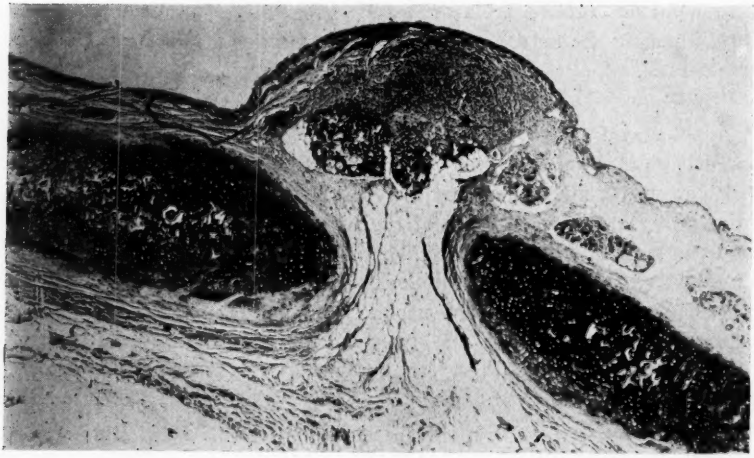

Fig. 2.—Photomicrograph of a metastatic nodule of melanosarcoma in the submucosa of a large bronchus.

lung had been made before his admission to the hospital and he was referred to this hospital for surgery. Twenty years prior to this time he had had pleurisy; at about the same time he had been treated successfully for gastric ulcer. Ten years later ulcer symptoms recurred but again responded to medical treatment. Otherwise he had always been well and strong. Examination of the chest on admission showed slight dullness and markedly diminished breath sounds, râles, and an occasional wheeze over the left upper lung, anteriorly and posteriorly. There was tenderness anteriorly over the third and fourth ribs. Roentgenograms of the chest showed a mass in the left upper lobe having the appearance of primary carcinoma with involvement of the pleura and the second rib (Fig. 3). A smear from an aspiration biopsy showed

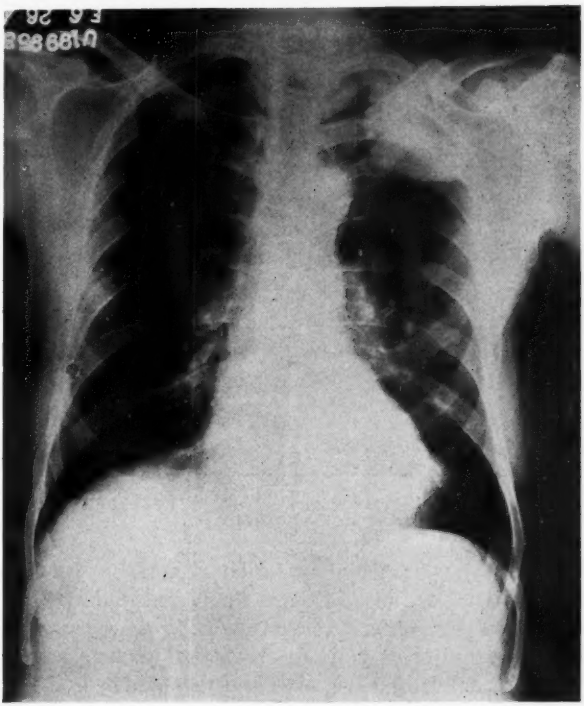

Fig. 3.—Roentgenogram showing a sharply defined mass in the left apex continuous with the chest wall. Autopsy revealed this to be metastatic from stomach.

tumor cells. A left upper lobectomy was performed in the belief that the tumor was primary in the lung. Pathologic examination of the surgical specimen revealed an undifferentiated carcinoma with extension to the periosteum of the rib but no invasion of the bone marrow or of the regional lymph nodes. Following operation he failed rapidly

and died on the second postoperative day from pneumonia in the right lung. Autopsy showed carcinoma of the stomach with extension to the pancreas and metastases to the regional lymph nodes. Microscopic sections of the resected lung were similar to those of the stomach and it was believed that the tumor in the lung had been secondary to that of the stomach.

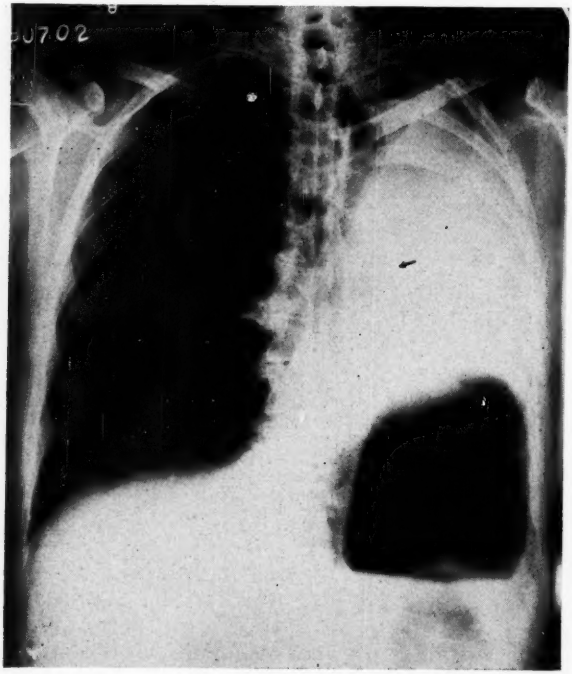

Fig. 4A.—Roentgenogram showing complete collapse of left lung due to intrabronchial tumor (arrow).

A case illustrating bronchial occlusion as well as hemoptysis produced by a metastatic tumor is that of a 42-year-old man who had a fibrosarcoma of the scrotum removed in 1938. One year later he developed a cough and chest pain and roentgenograms showed multiple sharply defined rounded masses scattered throughout both lung fields. Two months later his pulmonary symptoms were much more severe and included raising of bloodtinged sputum. Another roentgenogram showed complete collapse of the left lung and an x-ray film made with the Potter-Bucky technique demonstrated a smooth rounded mass within the left main bronchus (Fig. 4). Bronchoscopic removal of a large piece of the metastatic tumor produced marked relief in the patient's symptoms and almost complete expansion of the left upper lobe roentgenologically (Fig. 5). However, the tumors in his lung continued to

grow and he finally died of respiratory failure. Post-mortem examination showed complete occlusion of the left upper lobe bronchus for a distance of 4 cm. by a tumor plug and a similar occlusion of a second degree branch bronchus of the right lower lobe (Figs. 6 and 7).

In 1932, Vinson and Martin[1] and in 1936, Maytum and Vinson[2] described a pulmonary metastasis from hypernephroma with ulceration into a bronchus simulating primary bronchial carcinoma. Farrell[3] reviewed a group of seventy-eight cases seen at necropsy with pulmonary

Fig. 4*B*.—Same film with better photographic detail of bronchus. Arrows point to mass protruding into left main bronchus.

metastasis and found occlusion of a bronchus by metastasis to the wall uncommon. The patients in two of his cases had hemoptysis. Pendegrass and Hodes[4] also describe an unusual type of pulmonary metastasis from hypernephroma in which the patient developed a cough productive first of mucopurulent sputum and later of bloodstreaked sputum. The post-mortem report in this case does not state whether the bron-

chial tree was invaded. Bosse,[5] in a description of an autopsy case of pulmonary metastasis from teratoma of the testis in a patient who had no pulmonary symptoms other than dyspnea says, "Several smaller nodules (of tumor) bulged into the lumen of the bronchi of the right lung." Ewing,[6] in the 1940 edition of his work on neoplastic disease, does not discuss bronchial invasion by metastatic new growths. In 1941, Raine[7] reported a large solitary metastasis from an adenocarcinoma of the sigmoid invading and obstructing a major upper lobe bronchus.

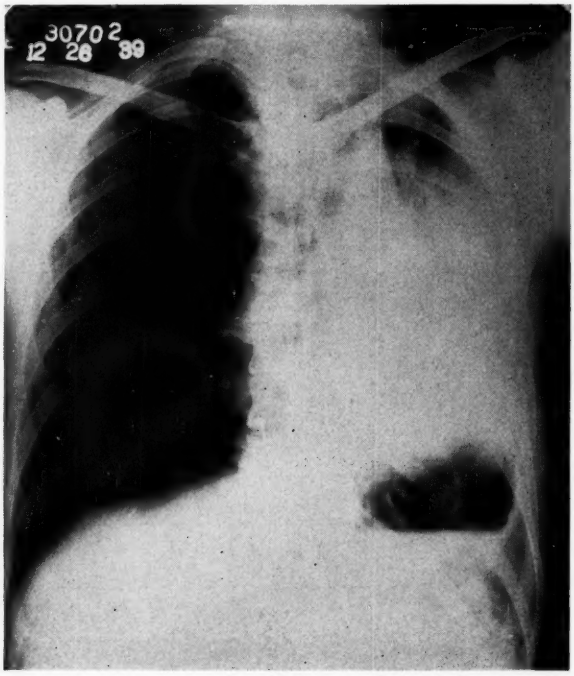

Fig. 5.—Same case as Fig. 4 showing partial aeration of left upper lobe following removal of portions of the intrabronchial tumor.

Our investigation covered 109 autopsies performed at the Massachusetts General Hospital between 1931 and 1940 (inclusive) on pa-

TABLE I

TYPE OF NEOPLASMS WITH PULMONARY METASTASIS

TYPE OF TUMOR	NO. AUTOPSIES WITH PULMONARY METASTASIS	NO. WITH ACTUAL BRONCHIAL INVASION	PER CENT BRONCHIAL INVASION	NO. WITH BLOOD-STREAKED SPUTUM
Carcinoma	98	14	14.3	3
Sarcoma	11	6	55.6	1
Total	109	20	18.3	4 (20%)

Fig. 6.—Photomicrograph at low magnification showing intrabronchial metastatic fibrosarcoma in right lower bronchus of same patient.

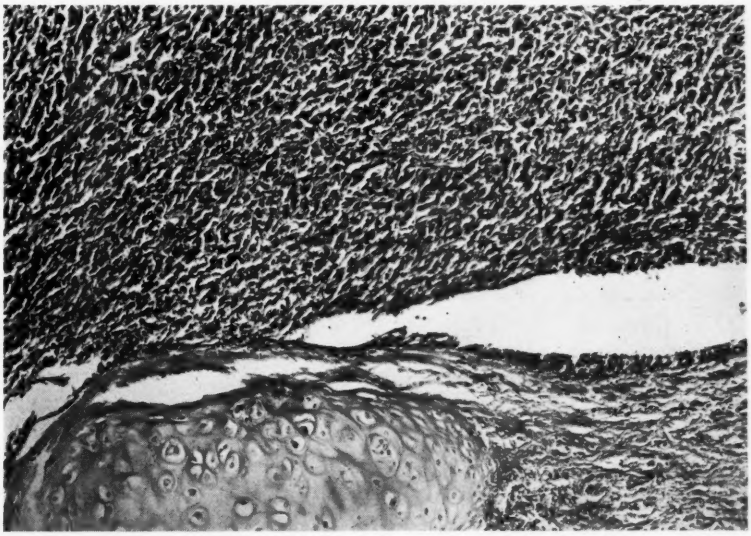

Fig. 7.—Photomicrograph at high magnification demonstrating the spindle-cell character of the tumor. Note normal ciliated respiratory epithelium where tumor is not attached.

TABLE II

PRIMARY SITES OF NEOPLASMS WITH BRONCHIAL METASTASIS

PRIMARY SITE	TYPE OF TUMOR	BLOODY SPUTUM	NO PULMONARY SYMPTOM	OBSERVED GROSSLY	BRONCHIAL INVASION (EXTENT)
Breast	Carcinoma		+	+	Infiltration of major bronchi from mediastinum; microscopically, tumor is present in wall of bronchi between cartilaginous plates
	Carcinoma				Microscopically, tumor occludes lumen of small bronchus as it grows both into it and also beneath the epithelium and around the cartilaginous plates
	Carcinoma				Microscopically, tumor cells almost completely block a medium-sized bronchus
	Carcinoma	+		+	Infiltration of all layers of a large bronchus from mediastinum
	Carcinoma				Microscopically, several medium-sized bronchi are extensively infiltrated with carcinoma and in one the mucosa has been replaced by tumor which partially obstructs the lumen
Stomach	Carcinoma	+		+	Extension from hilus to surround and invade the right upper lobe bronchus; small branches 3 mm. in diameter occluded by tumor
	Carcinoma	+		?	Lobectomy for supposed primary tumor performed; bronchi not examined but almost certainly involved
	Carcinoma		+		Microscopically, a small bronchus involved
Cecum	Carcinoma		+	+	A large bronchus, 7 mm. in diameter, ends in and is completely infiltrated by tumor
Rectum	Carcinoma		+	+	A second degree bronchus shows mucosal ulceration over an area 1.5 cm. due to extension of tumor from the lung parenchyma
Thyroid	Carcinoma			+	Numerous small metastatic nodules 2 to 5 mm. in diameter are present in the mucosa of trachea and bronchi
Mouth	Carcinoma		+		The mucosa of one small bronchus is partially replaced by neoplastic tissue
Prostate	Carcinoma		+		Microscopically, a small bronchus is practically destroyed by tumor
Cervix	Carcinoma		+		Microscopically, a bronchiole plugged with tumor
Bone	Osteogenic sarcoma				Microscopically, there is mucosal infiltration of secondary bronchus with plugging of lumen
	Chondrosarcoma			+	Secondary bronchi infiltrated and destroyed by tumor

TABLE II—CONT'D

PRIMARY SITE	TYPE OF TUMOR	BLOODY SPUTUM	NO PULMONARY SYMPTOM	OBSERVED GROSSLY	BRONCHIAL INVASION (EXTENT)
Thigh	Fibrosarcoma	?		+	Parenchymal extension into larger bronchi near hilus with mucosal involvement
Scrotum	Fibrosarcoma	+		+	Complete occlusion of main left upper bronchus for 4 cm. and of a 2 degree bronchus by tumor plugs
(?) Meninges	Melanosarcoma		+	+	Bronchus 9 mm. in diameter contains a 4 mm. tumor nodule protruding into lumen (Fig. 2)
Pharynx	Fibrosarcoma			+	Tumor masses in both upper lobes have penetrated the walls of the main bronchus and grown down in the form of fingerlike projections for 1 cm., completely occluding the lumen
Total		4	8	11	

tients with malignant growths primary elsewhere in the body which metastasized to the lungs. From this group, as demonstrated in Table I, there were twenty, or 18.3 per cent that showed definite infiltration of the bronchial wall or mucosa. Not included were those cases with complete or partial bronchial stenosis from extrinsic pressure due to tumor growth although it was noted that one of these cases did give a history of raising bloodstreaked sputum.

Of the twenty patients with definite bronchial involvement, fourteen were cases of metastatic carcinoma and six of sarcoma. While the number of sarcomas is too small to warrant drawing definite conclusions, it will be noted that the incidence of bronchial invasion in the sarcoma group is over three times that in the carcinoma group. This is probably due to the fact that the lung is the choice site of sarcomatous metastases and there would, therefore, be a greater chance for secondary involvement of the bronchi. Most of the autopsies in this series were performed in fairly routine fashion and no attempt was made to section every metastatic nodule to see its connection with a bronchus or to search very carefully throughout the bronchial tree for small metastatic mucosal nodules. Had this been done there is no doubt that the incidence of bronchial involvement would have been much higher. In nine cases bronchial invasion was discovered only microscopically from random sections. Here again if many more sections were taken for examination, more cases of bronchial involvement would have been found.

Since only four patients out of a group of twenty (20 per cent) raised blood, infiltration of a bronchial wall is not of itself sufficient to cause this symptom. In fact, 40 per cent of the entire group did not

complain of pulmonary symptoms in spite of the extent of the metastases. This is in sharp contrast to a group of 158 proved cases of primary bronchiogenic carcinoma, most of which had pulmonary symptoms and 51 per cent of which raised blood.

Although a large proportion of the cases of metastatic bronchial involvement reported in literature are hypernephromas, it is interesting to note that none of the four cases in our series of autopsies of hypernephroma with pulmonary metastases showed bronchial infiltration. One patient, however, who did not come to autopsy (and is not included in this analysis) had a pneumonectomy performed because of a tumor diagnosed microscopically as metastatic from a hypernephroma. No primary source has been found, although the survival period is now four years. The woman did not raise blood, but microscopic examination of the resected lung showed a tumor growth into the bronchial lumen. A second patient on whom a partial lobectomy was performed for a single metastatic tumor from hypernephroma showed no bronchial invasion. She was still alive and well nine years after operation (Barney and Churchill[8]).

There are two malignant neoplasms that may produce bronchial infiltration that were not included in this series: Malignant lymphoma and primary carcinoma of the esophagus. In both of these lesions bronchial involvement is usually by direct extension from mediastinal tumor masses rather than by metastasis. In lymphoma there is the further problem of whether a given nodule of tumor is to be regarded as an independent focus of neoplasia or as a metastasis in the ordinary sense. Three cases in which the patients had definite ulceration or infiltration of a bronchus, however, were found among the autopsies reviewed. Primary cancer of the esophagus was found to have invaded or ulcerated a major bronchus in five cases, in two of which there was hemoptysis. This bronchial involvement was by direct extension from the primary lesion and here also this does not constitute a true metastasis.

SUMMARY

1. Necropsies performed at the Massachusetts General Hospital over a ten-year period on patients with metastatic pulmonary neoplasm were investigated in order to discover the incidence of those in whom actual invasion of the bronchus occurred.

2. In 109 cases of pulmonary metastatic tumor 18.3 per cent showed bronchial invasion. In addition to these, three patients with malignant lymphoma showed bronchial invasion. It is believed that the incidence would have been much higher if especial attention were paid to the gross examination of the complete bronchial tree and if more sections were taken for microscopic study.

3. Detailed analysis was made in twenty cases in which infiltration of the bronchial tree was found and the following observations were made:

(a) The neoplasm in fourteen was carcinoma; in six, sarcoma.
(b) Four patients (20 per cent) raised bloodstreaked sputum.
(c) Nine patients (40 per cent) had no pulmonary symptoms.

CONCLUSIONS

1. Metastatic pulmonary neoplasm may simulate a primary tumor.

2. In a patient suspected of having pulmonary neoplasm, the raising of bloodstreaked sputum cannot be considered as pathognomonic of a *primary* tumor.

3. But the raising of blood in a case of metastatic pulmonary neoplasm is rare. In the present study, it occurred in only 4 out of 109 cases.

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EXTRAPLEURAL PNEUMOTHORAX

Report of Thirty-Eight Cases

John G. Russo, M.D.
Wales, Wis.

ALTHOUGH the operation of extrapleural pneumothorax was first performed by Tuffier, in 1910, the gradual development and institution of this form of collapse therapy for the treatment of pulmonary tuberculosis has been more fully recognized and recorded by numerous authors since 1936. Impressed with the usefulness and encouraging successful reports of other authors, this surgical measure for producing cavity closure and negative sputum was adopted at the Wisconsin State Sanatorium.

The following report is a result of our experience with thirty-eight patients on whom extrapleural pneumothorax was performed. The surgery was done by one individual, Dr. J. W. Gale, of the State of Wisconsin General Hospital. The period of treatment extended from August, 1938, to July, 1942. The operation was performed forty-six times on the thirty-eight patients.

AGE GROUP

In the group studied there were sixteen males and twenty-two females. The youngest patient was 21 years of age. Five of the patients were in the latter part of the sixth decade, and the two oldest patients were 63 and 67 years old, respectively.

SELECTION OF CASES

The patients selected for this type of surgery were those in whom bed rest and previous type of collapse therapy had proved unsuccessful.

In a large proportion of cases pneumothorax could not be induced. In the cases in which intrapleural pneumothorax was induced the collapse was unsatisfactory and had to be abandoned because of multiple large, dense adhesions present which could not be severed. Diaphragmatic paralysis was also given adequate trial.

As previously mentioned, the disease in all of the cases was far-advanced. The duration of disease, prior to surgery, averaged five years.

Extrapleural pneumothorax was selected for the group with bilateral lesions because cavitation was present in both apices. In the group in which thoracoplasty had been performed and with disease in the contralateral apex, the operation was chosen because of the selectivity of collapse, thus preserving as much lung tissue as possible.

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In the older patients (55 to 67 years of age) this measure was primarily chosen because of decreased danger from shock and a preference for a single-stage operation.

Extrapleural pneumothorax was performed instead of thoracoplasty for "balloon tension type" of cavity in three cases. Only one yielded to the operation with closure of cavity and sputum conversion.

In our early adoption of extrapleural pneumothorax some of the patients selected had a hopeless outlook. Thoracoplasty was possible in a few of the cases but extrapleural pneumothorax seemed less trying and better suited to the individual. As experience with this procedure developed we have narrowed down our indications. At present, we no longer use the operation in hopeless cases and still consider thoracoplasty preferable to extrapleural pneumothorax.

RESULTS

Thirteen patients were discharged. Twenty-one are still in the sanatorium receiving treatment and four are dead. Thirty of the patients had unilateral extrapleural pneumothorax and eight had bilateral. Of the bilateral group one died. There were four patients with a thoracoplasty on the contralateral side at the time of operation and of this group one died.

Including the four deaths, sputum conversion to date reveals: nineteen cases, or 50 per cent, negative on the basis of concentrated sputum study and guinea pig inoculation of gastric contents; three cases repeatedly negative to concentrated sputum examination but positive to guinea pig inoculation of gastric contents. This makes a total of twenty-two cases, or 58 per cent, considered negative, which includes the three positive to guinea pig inoculation, but past observation indicates that these will most likely turn negative. Excluding this group in which complete conversion as yet has not taken place, the per cent figure of sputum conversion is 50 as revealed by the most exacting methods. Sixteen cases, or 42 per cent, are positive.

Twelve patients, or 32 per cent, developed extrapleural empyema. Of these, eight developed extrapleural empyema of the pure type and four developed a mixed type. Those of the pure type were treated by repeated aspirations, irrigations, and instillation of azochloramide. In six cases empyemas cleared and two are still undergoing treatments. Of the six, five were converted to oleothorax and in the other the extrapleural pneumothorax space was successfully obliterated by a thoracoplasty. It is interesting to note that this particular patient, a woman 54 years of age and one of the early extrapleural pneumothorax cases, was the only one in which expansion of the underlying lung was attempted. We were successful in obtaining partial re-expansion. However, a tuberculous empyema developed twenty-seven months after the date of surgery. To date this patient's sputum is negative to concentrated smear and guinea pig inoculation of gastric contents. It is pos-

sible that we may do a thoracotomy in one of the mixed empyema cases in the near future.

After maintenance of extrapleural pneumothorax for an adequate period of time, twenty-six extrapleural spaces, including bilateral cases, were converted to oleothorax using mineral oil, and six spaces were obliterated by thoracoplasty.

DISCHARGED CASES

The patients in three cases were discharged quiescent and ten apparently arrested. Questionnaires were sent to all discharged patients and the latest information received revealed: Five patients doing light work averaging eight hours a day, three patients doing housework, two at the state rehabilitation camp working part time, one attending vocational school, and two not working. Of the latter, one is a bilateral case and the other developed clear straw-colored fluid underneath the oil in the extrapleural space. The fluid was removed and replaced by oil and the culture studies of the fluid revealed no tubercle bacilli. Incidentally, this feature had occurred in other cases and the fluid was found negative to guinea pig inoculation. The fluid occurred with no marked symptoms and was detected by routine fluoroscopic studies and x-rays.

DEATHS

Post-mortem examinations were performed on the four patients who died.

The first patient, a man, 32 years of age, died sixteen months following the operation, after developing a mixed infection empyema with bronchoextrapleural fistula and progression of disease in the contralateral lung. Death occurred one year after development of empyema.

The second patient, a woman, 23 years of age, died sixteen months after surgery from sepsis and extension of tuberculosis involving both lungs. The patient was making satisfactory clinical progress after the establishment of bilateral extrapleural oleothorax when she developed a mixed infection empyema on the right side with bronchoextrapleural cutaneous fistula. Death occurred one year after development of empyema.

The third patient, a woman, 24 years of age, diabetic, Indian-Irish extraction, died from hemorrhage nineteen months following operation. The bleeding originated from progression of disease on the unoperated side.

The fourth patient, a woman, 29 years of age, died five months after surgery. She had a thoracoplasty on the right side and developed a "balloon type" of cavity in the left apex. She was fighting a losing battle and extrapleural pneumothorax was done to stem the tide. Despite the effort, steady progression of the disease occurred. Post-mortem examination revealed the cavity to be smaller and that death resulted from right-sided heart failure.

SURGICAL REMARKS

Morphine sulfate and scopolamine hydrobromide were used routinely two hours before surgery and each patient received intravenous fluids during surgery. Cyclopropane anesthesia was used in all cases. To date there have been no anesthetic complications.

In one case not included in this report, due to extensive adherence of the parietal pleura to the apex of the chest wall, it was inadvisable and dangerous to proceed with the operation.

There was a case in which the parietal pleura was inadvertently opened during the process of dissection and an extraintrapleural pneumothorax resulted which was maintained five months as a combined space and sputum conversion took place, but because of the obliteration of the space the treatment had to be discontinued.

In the third and last case of "balloon tension type" of cavity partial loss of the extrapleural pneumothorax space resulted coincident with marked subcutaneous emphysema following the first injection of air which was done the afternoon of surgery. Three months later a revision extrapleural pneumothorax was attempted but it failed. A thoracoplasty was performed and atelectasis occurred. To date cavity closure is uncertain and there is progression of disease in the other lung.

POSTOPERATIVE CARE

Oxygen by nasal catheter, intravenous fluids, and mild sedatives are used routinely after surgery. Upon returning to bed the patients were placed in a recumbent position. After reaction from the anesthetic was complete, the head of the bed was elevated six inches. It is important that postoperatively the patient be studied very closely or the loss of the extrapleural pneumothorax space may occur. By observing the size of the extrapleural space at the time of surgery it can be determined whether it may be necessary to inject air the afternoon of surgery or the following morning. Periodic fluoroscopic study is important and renders valuable information. It has been our practice to remove the serosanguineous fluid soon and keep the space dry. The pressures in the space are maintained near atmospheric early. Positive pressures as required should be established within the first two weeks, during which time the operator should seek to maintain the optimum size of the space desired. Attempts to either enlarge or reduce the space at a later date are undesirable if not impossible.

After the sputum is negative, conversion to oleothorax or thoracoplasty as indicated may be done. We prefer to have the space dry and stable before attempting oleothorax. In establishing extrapleural oleothorax the essential precautions in intrapleural oleothorax are observed. The individual is given a trial test dose of mineral oil and the amount is gradually increased as tolerated by the patient. I have been

impressed with the beneficial results of marked restriction of exercise during establishment of extrapleural oleothorax.

SUMMARY

A report of thirty-eight patients treated by extrapleural pneumothorax has been reviewed. The results are summarized in Table I.

TABLE I

DATA OF 38 CASES

	NO.	PER CENT
Discharged	13	34
Still in sanatorium	21	55
Dead	4	11
Bilateral cases	8	21
Thoracoplasty present on contralateral side at time of surgery	4	11
Sputum conversion to date	22	58
Extrapleural empyema	12	32
Conversion to thoracoplasty	6	16
Conversion to oleothorax including bilateral cases	26 pockets	

CONCLUSIONS

Extrapleural pneumothorax has been selected for individuals for whom a thoracoplasty originally was not recommended. We feel that after cavity closure, loss of toxicity and physiologic adjustments that have taken place with extrapleural pneumothorax, the patient is then rendered a suitable candidate for thoracoplasty. Thoracoplasty is a permanent form of collapse whereas extrapleural pneumothorax is a quasipermanent form of collapse. The complications of extrapleural pneumothorax are severe enough to not choose the procedure in preference to thoracoplasty if the latter is feasible. Thoracoplasty will reduce the incidence of empyema.

Oleothorax is to be used only in cases in which it is impossible to do a thoracoplasty at a later date because of age, low vital capacity, poor general condition, or because the patient is unwilling to submit to thoracoplasty.

This study of extrapleural pneumothorax suggests that as a collapse measure for the treatment of pulmonary tuberculosis it still holds a place in thoracic surgery. Its indications are being narrowed down from time to time, making the operation of distinct benefit in a relatively small number of individuals.

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EXPERIMENTAL PRODUCTION OF INSUFFICIENCY AND STENOSIS OF THE HEART VALVES IN DOGS*

M. M. Shaw, M.D., W. E. Adams, M.D., R. A. Rasmussen, M.D.,
L. S. Hrdina, and H. G. Aronson, M.D.
Chicago, Ill.

THE rapid advance of thoracic surgery during the past twenty-five years has been the direct result of increased knowledge of the physiology of the intrathoracic organs, improvement of diagnostic methods, development of positive pressure anesthesia, and a better appreciation of the problem of open pneumothorax. This better knowledge of the normal cardiorespiratory physiology and the disturbances brought about by changes in intrathoracic pressure and obstruction to the blood flow to and from the heart have permitted us to go farther in the study of heart physiology. This progress has brought us to the study of complex pathologic processes of the heart, namely valvular stenosis or insufficiency and vegetations, pericardial adhesions, and disturbances of the heart caused by tamponage and patent foramen ovale together with other lesions.

A thorough review of the experimental production of valvular lesions of the heart has been presented by Cutler, Levine, and Beck[1] along with their personal contributions. The main stimulus for these studies has been the need of a satisfactory operation for the alleviation of mitral stenosis. Although considerable success has attended the production of valvular lesions in animals, especially by Cushing and Branch,[2] Cutler,[3] Beck and Cutler,[4] and Allen and Graham,[5] the results of its clinical application have been rather discouraging.

The object of this study was to seek a better understanding of the fundamental derangement of the normal cardiorespiratory function brought about by stenosis and insufficiency of the heart valves. This report will present the technique developed in the experimental production of stenosis or insufficiency of the tricuspid, the mitral and the pulmonic valves.

In this laboratory it has been shown that cessation of blood flow through the heart was well tolerated for at least five minutes.[6] Cardiac activity remained regular for as long as nine minutes with complete cessation of blood flow to the right heart, and the heart functioned normally after the obstruction to the flow had been released.

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Of the large vessels connected with the heart, obstruction of the superior and inferior vena cava and the azygos was by far the best tolerated. With this information available, a method of approach to the cardiac valves under direct vision presented itself.

EXPERIMENTAL PROCEDURE

Dogs were used exclusively throughout the study. After preoperative sedation with morphine sulphate and atropine sulfate, operations were made using positive pressure intratracheal ether anesthesia.[7] The approach was through the right or the left fifth intercostal space, a rib resection not being necessary for adequate exposure. The blood flow to the heart was controlled by placing ligatures of black linen thread about the inferior and superior vena cava and the azygos vein with the aid of an aneurysm needle. The proper tension applied to these sling ligatures shut off the blood flow to the heart (see Fig. 1).

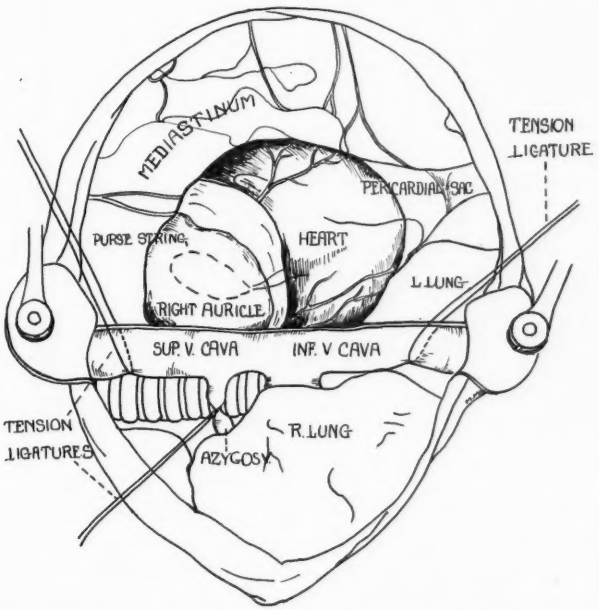

Fig. 1.—Diagrammatic illustration of exposed organs and structures in the production of lesions of the tricuspid valve. Note tension ligatures about the venae cavae and a purse-string suture in the wall of the right auricle.

In the tricuspid procedure, after opening the pericardium longitudinally, a purse string of black silk was placed in the wall of the right auricle avoiding the appendage. At this stage of the procedure, tension was applied to the ligatures about the vessels. A few seconds were allowed to pass so that the heart might empty itself of the major

portion of the blood that it contained. The auricle was then entered through the purse string. Allis forceps were applied to either margin of the sectioned auricular wall and used for traction. Visualization was secured by the use of suction and an endoscopic light (see Fig. 2). The approach permitted the excision of one or more cusps of the tricuspid valve forming an insufficiency or permitted the placing of a suture into the valve cusps or a ligature about the chordae tendineae, thereby creating a stenosis.

The pulmonic valve was reached by going through the right ventricle near the interventricular septum. Interrupted approximating sutures were used in the ventricular wall instead of the purse-string suture. With the aid of a dural hook, the free margin of a pulmonic leaflet was grasped and cut away with scissors or a septum punch, thus forming a pulmonic insufficiency.

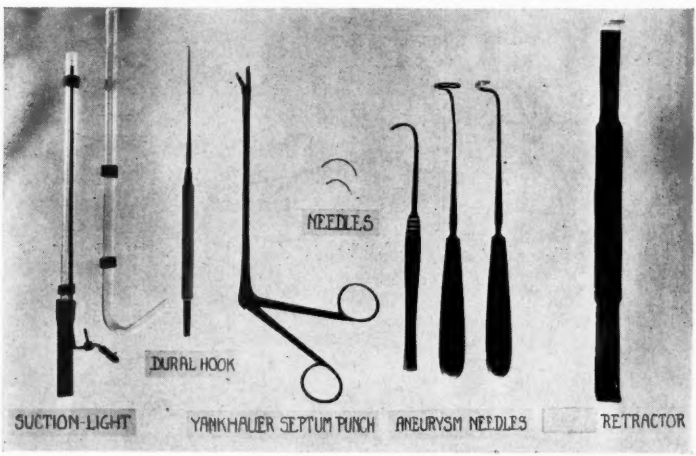

Fig. 2.—Instruments and suction apparatus used in the production of valvular lesions.

In surgery of the mitral valve the approach was through the left auricular appendage, the left chest having been first entered through the fifth interspace. In the earlier experiments the superior vena cava was reached by blunt dissection through the mediastinum into the right pleural cavity and a sling ligature was placed about the vessel. The azygos was secured with more difficulty through the same opening in the mediastinum by retracting downward and slightly forward on the base of the heart with a special retractor (Fig. 2). A sling was then placed about the azygos vein with a right angle aneurysm needle. This ligature was tied immediately, thus cutting off the azygos vein permanently, or tension was applied to the sling ligature about the vein to stop the blood flow and the ligature later released. The inferior vena

cava was reached by entering the right pleural cavity through the lower mediastinum and a sling placed about it, again using an aneurysm needle. Recently the procedure has been altered so that sling ligatures are placed only about the superior and the inferior vena cava. After having thus secured control of the blood supply to the heart, the pericardium was opened parallel to the phrenic nerve. The tip of the left auricular appendage was caught in a clamp and the heart was rotated slightly upward and to the right, thus exposing the base of the appendage. A suture of double black linen was passed through the base of the appendage and tied. Tension was then applied to the ligature about the superior vena cava. Only a slight amount of tension was applied to the inferior vena cava, thus only partially cutting off the blood flow through the vessel. The wall of the auricular appendage was incised, the edges were retracted with Allis forceps, the valve leaflets grasped with a tooth forceps or sharp curved aneurysm needle, and the leaflets sutured together with a needle, or a portion of the leaflet was excised thereby creating, respectively, a stenosis or an insufficiency. After the production of the lesion the entire auricular appendage was tied off at its base with the previously mentioned stay suture.

After production of any one of the above valvular lesions the incision into the myocardium was closed, the sling ligatures quickly released (the superior vena cava being released first), the heart observed for any visual or palpable change, the pericardium closed with interrupted sutures, the lungs reinflated, and the chest wound repaired. The time was noted during which the circulation had been obstructed, and the blood loss during the intracardiac manipulation was measured.

Two techniques were used in creating the stenotic lesions. One method consisted of suturing the base of one leaflet to another; the other was the passage of a ligature about the chordae tendineae of opposite valves. The valvular insufficiencies were created by sectioning one or more valve leaflets.

Points of procedure that were of great value in facilitating the operation were: (1) Sling ligatures for control of blood flow; (2) the use of suction and an endoscopic light for visability (except in mitral valve lesions); (3) the use of a dural hook and special small retractors for ease in the removal of the valves; (4) the use of an aneurysm needle or a specially constructed needle for applying the ligatures to the valves in creating the stenosis; and (5) a purse string for rapid closure of the right auricle, and the transfixation suture for the left auricular appendage.

The average amount of blood loss during surgery involving the heart valves was 100 or 200 c.c. with a maximum of 350 c.c. in the left heart operations. We have given blood transfusions to replace this loss during operations upon the mitral valve. The time actually spent in work-

ing upon the valves varied from one to three minutes or an average of one and one-half minutes. A well-planned procedure with attention to details and minimal operating time greatly reduced the mortality.

RESULTS OF EXPERIMENTS

Table I presents a summary of the results of cardiovalvular lesions produced in thirty-five dogs. It has been much easier to work upon the right side of the heart than upon the left. More extensive lesions have been produced with greater ease, with less altering of function, and with lower mortality. In producing mitral valve lesions, more profound changes occurred in cardiac function, the technical difficulties were greater, and the mortality was higher. Of the two lesions of the mitral valve, it was more difficult to produce a stenosis than an insufficiency.

TABLE I

SUMMARY OF RESULTS OF CARDIOVALVULAR LESIONS PRODUCED IN 35 DOGS

VALVULAR LESION	NO. OF LESIONS	DIED	SACRIFICED	DURATION
Tricuspid insufficiency	12	3	9	1 day to 14 months
Tricuspid stenosis	8	2	3*	14 days to 11 months
			3	
Pulmonic insufficiency	3	1	2	18 days to 11 months
Mitral insufficiency	8	6	2*	10 minutes to 3 months
Mitral stenosis	6	6	0	2 days to 25 days
Total lesions	37	18	19	

*Alive and apparently well.

Earlier in the development of technique, a very high mortality prevailed with operations upon the mitral valve. Sufficient tension on the sling ligatures about the inferior and superior vena cava and the azygos was applied to shut off the blood flow completely. It was observed that air entered the coronary arteries through the opened left auricle and ventricle. Following the coronary air embolism the heartbeat decreased in force and rate of contraction and went into a fatal generalized fibrillation. Many acute experiments were performed, which are not recorded here, in an attempt to prevent the coronary air embolism. Success has attended surgical procedures upon the mitral valve when little or no tension was placed upon the sling ligature about the inferior vena cava, thus permitting a free flow of blood out through the opening in the left auricle and preventing air from entering the left ventricle and progressing on to the coronary arteries. This was further facilitated by having the animal placed well over on the right side. An additional precaution to prevent coronary air embolism was to keep the valve bathed in blood during the operation rather than to retract it up into the opening in the left auricle.

Because of the loss of blood encountered in producing a mitral stenosis, citrated whole blood transfusions were employed during the

operation. The blood was introduced into the jugular vein; a rapid flow was permitted at the moment of closing the left auricular appendage by relaxing the sling on the superior vena cava.

VALVULAR LESIONS

The changes resulting from the valvular lesions were more marked when the lesion was on the left side of the heart. Few animals have survived the production of a mitral lesion. One dog with a mitral steno-

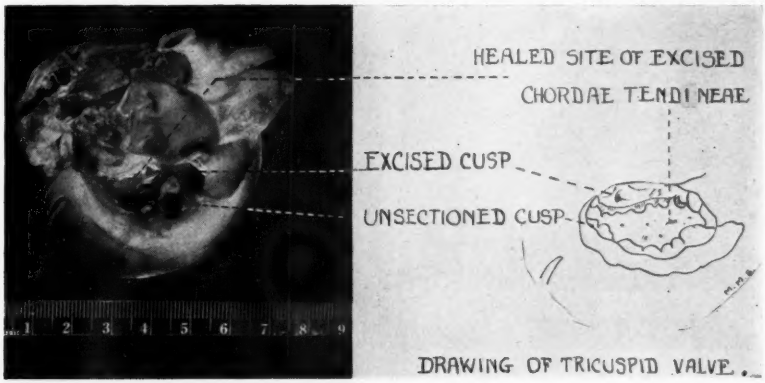

Fig. 3 (Dog 652).—Tricuspid insufficiency by excision. Note the one completely excised cusp and intact cusp, also healed site of excised chordae tendineae. Sacrificed after two months.

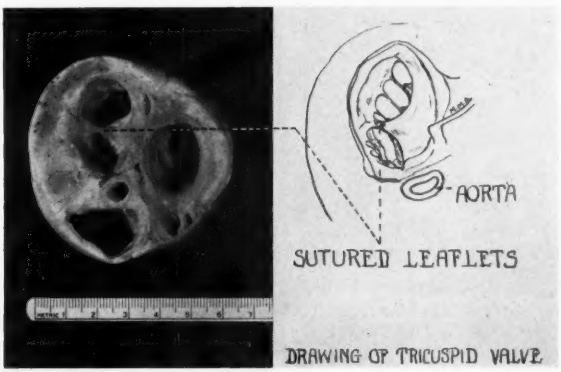

Fig. 4 (Dog 67).—Tricuspid stenosis by suture. Note sutured leaflets and funnel-shaped deformity. The openings measure 15 by 7 mm. and 10 by 4 mm. Sacrificed after 11 months.

sis lived twenty-five days (Dog 150) and another animal with a mitral insufficiency lived three months (Dog 90). Dogs having a tricuspid lesion have survived and appeared healthy as long as fourteen months (Dog 885).

TRICUSPID INSUFFICIENCY OR STENOSIS

When the heart was observed directly after a tricuspid insufficiency or stenosis, the right auricle and especially its appendage was markedly dilated. This was followed by a temporary dilatation of the right ventricle and subsequently by a dilatation of the entire heart. There was a distinct thrill palpable through the heart wall over the location of the valve (Figs. 3 and 4).

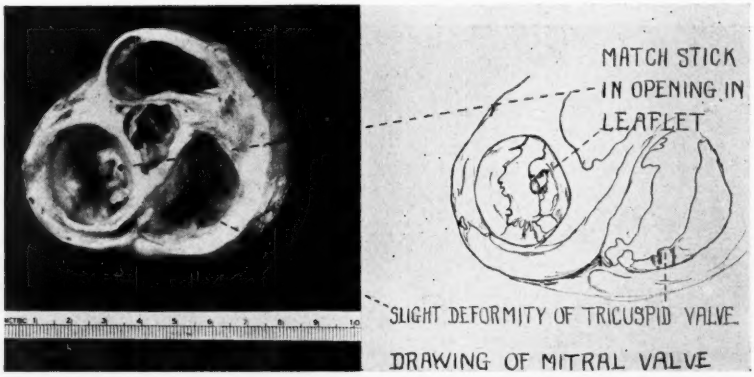

Fig. 5 (Dog 11).—Mitral insufficiency by incision plus tricuspid stenosis by suture. Note the matchstick in opening in mitral leaflet; slight puckering of tricuspid leaflet by suture in the valve. Sacrificed three months after first operation.

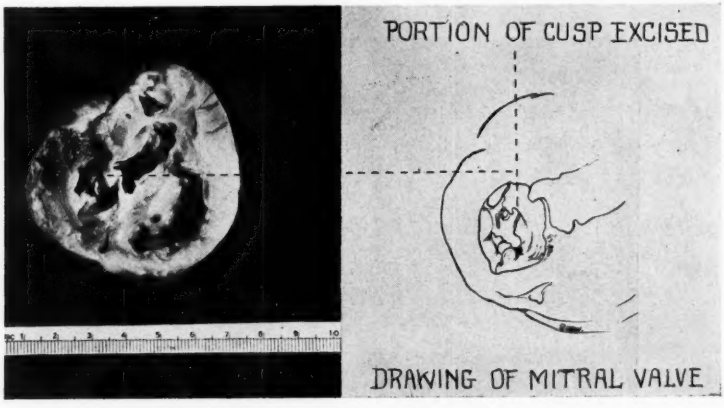

Fig. 6 (Dog 152).—Mitral insufficiency by excision of a portion of mitral leaflet. Note defect in the leaflet. Dog died eighteen hours after the operation.

PULMONIC INSUFFICIENCY

In the pulmonic insufficiency lesions the right ventricle bulged and a thrill was distinctly palpable (Fig. 8). One entire cusp was removed in one animal without the production of cardiac decompensation. This dog

(No. 258) remained healthy and active until sacrificed twenty-two months after operation.

MITRAL INSUFFICIENCY

After the production of a high-grade mitral insufficiency, the pulmonary conus in the right ventricle bulged immediately. The right ventricle became dilated and a thrill was palpable. The pulmonary vessels were tense and greatly distended by the increased pressure. In some instances the dilatation was so marked that the pericardial sac became distended and the space obliterated. If the mitral insufficiency was slight or of moderate degree, the dilatation of the cardiac chambers was not so striking. The heartbeat recovered its rate and some of its force within two to five minutes if the experiment was successful and the animal survived (Figs. 5 and 6).

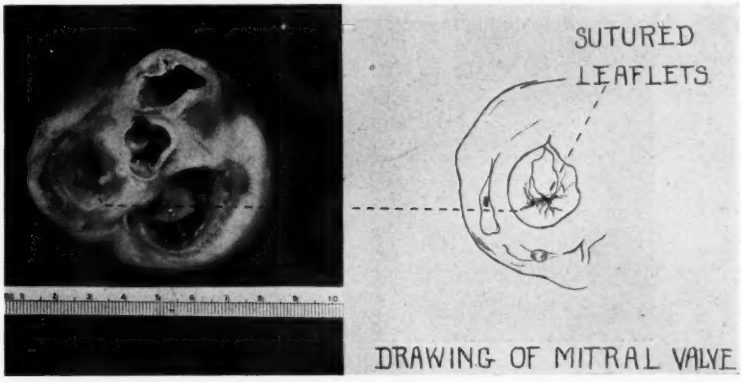

Fig. 7 (Dog 232).—Mitral stenosis by suture. Note the high degree of stenosis produced by suturing the two leaflets together. Dog lived five minutes.

MITRAL STENOSIS

In a mitral stenosis of marked degree the left ventricle was observed to become smaller and then gradually resume its normal size as the heart recovered from the operative trauma and the chamber filled with blood. The pulmonary veins and the left auricle bulged with increased pressure. Although in some instances where a seemingly adequate stenosis was produced and a definite thrill was palpated, the changes in the heart have been slight. Evidently the lesion did not produce a functional stenosis (Fig. 7).

In summary, therefore, after recovery of the animals the external examination and the findings upon examination of the heart were as follows: Definite murmurs were demonstrable in all animals but there was no obvious evidence of cardiac decompensation. The animals were

active. There was no dyspnea, no edema of the conjunctivae, and no marked enlargement of the jugular veins. There was no edema of the forelegs, except in one instance (Dog 299) where there was a generalized anasarca associated with two cardiovalvular lesions and a complicating thrombosis of the coronary sinus. However, in the pulmonic insufficiencies and the long-standing tricuspid stenosis (Dogs 522 and 523), the jugular veins were more distended.

ASSOCIATED PATHOLOGIC FINDINGS

The purse-string suture in the right auricular wall, when tied, encroached upon the size of the cardiac chamber. In a similar manner, ligation of the left auricular appendage decreased the volume of blood within the auricle but to a somewhat less degree. If the purse string was large, if an accidental cutting of the purse string occurred with subsequent resuturing, or if the auricular wall tore, the capacity of the

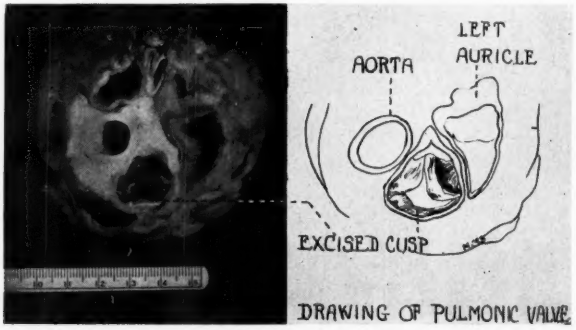

Fig. 8.—(Dog 258).—Pulmonic insufficiency by excision. Note the healed ridge of the excised pulmonic leaflet. The other two leaflets are intact. Diffuse fibrous obliterative pericarditis.

auricle was further compromised. The reduction of the size of the auricle did not seem to influence the chance of survival of the animal. Post-mortem studies of longstanding lesions suggest that the auricles have again enlarged to their preoperative size. The suturing of the right ventricle in tricuspid insufficiency reduced the size of that chamber only slightly. The ligated left auricular appendage usually contained a thrombus which became organized. This seldom caused trouble. However, in one instance a thrombus developed at the point of incision into the right auricle and the dog (No. 29) became seriously ill. A small mural thrombus was present in the left auricle together with a thrombosis of the coronary sinus in the only dog (No. 299) with decompensation. A thrombus attached to the mitral valve and the wall of the left ventricle was observed in Dog 150. The valvular lesions showed smooth healed surfaces or presented numerous small white nodules along the cut edge of the valve. The point of excision of the

chordae tendineae showed as healed thin grayish scars. One long chordae tendineae in a tricuspid insufficiency that was free and constantly bathed with blood showed a yellow, firm, calcific deposit on its surface (Dog 718).

The postoperative condition was studied by fluoroscopy and by x-ray examination. There was no evidence of cardiac decompensation or cardiac enlargement following any of the valvular lesions. An occasional infection of the chest wound was observed. There was no instance of an infection of the heart wound and no postoperative hemorrhages. The pericardium healed satisfactorily with an occasional adhesion along the incision.

Focal and diffuse fibrous obliterative pericarditis was found in some instances (Dogs 885, 334, and 718), probably the response to surgical trauma or the irritation of sutures. Although thrombosis occurred once, and three instances of mural thrombi were noted, there has been no evidence of embolism. Temporary dilatation of cardiac chambers occurred, but whether or not subsequent hypertrophy followed cannot be stated accurately at this time. Some specimens suggested cardiac hypertrophy by the thickening of the ventricular walls, the enlargement of the columnae cornae and the papillary muscles, and the shortening of the chordae tendineae. There were instances of focal pneumonia (Dog 384) and fibrinous pleuritis.

DISCUSSION

Our work was well under way and many of the difficulties of the technique and the hazards of the procedure had been understood and solved when the studies of Haecker[8] were encountered. He performed thirteen experiments in dogs producing valvular defects, septal defects, and resections of part of the superior vena cava and the thoracic aorta. He reported that no dog in which the cardiac chambers had been opened, lived more than a few minutes. One dog lived twelve days, in which a mitral insufficiency had been produced by stitching a mitral leaflet to the left ventricular wall without opening any heart chamber. Another dog lived twelve days after having a constricting suture placed about the aorta at the location of the aortic valves.

O'Shaughnessy[9] reported an elaborate method of perfusion of the brain with oxygenated Amberson-Hober 5 per cent solution of hemoglobin in Ringer's solution to prevent cerebral ischemia. This we have not had to do and our animals have continued to function and behave normally in so far as we were able to observe. In operations upon the right side of the heart, blood transfusions were not necessary but in more recent work upon the left side of the heart in the production of mitral stenosis we have employed citrated whole blood transfusions to replace blood loss.

It was astonishing to observe the degree of surgical trauma that a dog's heart would tolerate, namely, the incision, the handling, the interrupted blood flow, the excision of the valve leaflets or the suturing of two leaflets followed by the closure of the surgical wound. When the changes wrought by altered function and some degree of hemorrhage are added, the ability of the heart to withstand the procedure is all the more remarkable.

It has been our observation, however, that if too much tension is applied to the sling ligatures to control the blood flow, the heart will tolerate the procedure very poorly. Following such excessive tension upon the sling ligatures the heart sometimes went into ventricular fibrillation. We believe that cessation of blood flow through the heart is important for direct visualization of the right heart valves thus permitting more rapid and accurate production of the desired lesion.

This method of producing cardiovalvular lesions under direct vision is of significance because a definite planned lesion may be produced. It affords an opportunity for studying the effects of experimental stenosis and insufficiency of the cardiac valves. With this information at hand one may be able to evaluate more accurately the risks of surgical procedures in patients having cardiovalvular disease. Furthermore, with this knowledge, more successful active surgical intervention in valvular disease may be attempted.

SUMMARY

Extensive excisions of the tricuspid valve leaflets in dogs have been made. A moderate degree of stenosis of the tricuspid valve has also been accomplished by either suture or ligature. Both lesions were tolerated very well. Insufficiency of the pulmonic valve was also produced and likewise was well tolerated. Mitral stenosis and insufficiency have been created by suture and sectioning of the valve cusps respectively. The right heart will tolerate more surgery and more extensive lesions than the left heart. It is more difficult to produce lesions with survival of the animal when working on the mitral valve.

Any portion of the myocardium must be handled with care, first because the muscle is friable and second because irritation of the sensitive pericardium may initiate auricular or ventricular fibrillation.

Following the demonstration of the fact that a normal dog's heart will tolerate an interrupted blood flow for at least five minutes, direct visualization of the heart valves while operating upon them has been accomplished.

Further detailed studies of the effect of the above lesions are being carried out in collaboration with Dr. Emmet Bay. These include changes in cardiac output, blood pressure, intracardiac pressure, the electrocardiogram, and changes in the cardiac shadows as determined by the fluoroscope and the x-ray.

PROTOCOLS

Dog 258.—On Jan. 17, 1939, a pulmonic insufficiency was created by excising a leaflet of the pulmonic valve. After giving a normal healthy dog a preoperative dose of morphine and atropine an operation was made under intratracheal positive pressure ether anesthesia. The incision was made in the fifth intercostal space on the right side. The superior vena cava was isolated and a sling ligature of black linen thread was placed about the vessel, using an aneurysm needle. The azygos was identified, isolated, and ligated. Similarly a sling ligature was placed about the inferior vena cava. The pericardium was opened longitudinally. The approach to the pulmonic valves was through the right ventricle near the interventricular septum. A suture of double black linen was placed in the apex of the heart for traction. Four

TABLE II

RESULTS OF TRICUSPID INSUFFICIENCY IN 12 DOGS

DOG	LESION	DURATION	DIED	SAC.	MUR.	GROSS PATHOLOGY	REMARKS
718	Tri. ins.	2½ mo.		+	+	1 cusp removed	Firm calcific deposit on sectioned chordae tendineae; pericarditis
727	Tri. ins.	8 days	+			2 cusps removed	Died of pulmonary edema and early pneumonia
652	Tri. ins.	2⅕ mo.		+	+	1⅔ cusps removed	Right heart dilated
26	Tri. ins.	12 days	+		+	1⅔ cusps removed	Fibrinous pleuritis with effusion; fibrinous pericarditis with effusion; thrombus atelectasis
220	Tri. ins.	1 day		+	+	½ cusp removed	Large mural thrombus
699	Tri. ins.	2 days		+	+	2⅔ cusps removed	No cardiac decompensation
885	Tri. ins.	14⅓ mo.		+	+	Almost 2 cusps of tricuspid valve removed	Healed pericarditis; died at second operation (mitral valve)
237	Tri. ins.	8 mo.		+	+	1½ cusps removed	No cardiac decompensation
293	Tri. ins.	9 mo.		+	+	1 cusp removed	No cardiac decompensation
244	Tri. ins.	1 hr.	+		+	1 cusp removed	No cardiac decompensation
181	Tri. ins.	12 mo.		+	+	⅓ of two cusps removed	Died at second operation of coronary air embolism; mitral ins. attempted
299	Tri. ins. Mitral ins.	12⅔ mo.		+	+	2 cusps removed; T-shaped tear in mitral cusp	Thrombosis of coronary sinus, marked edema, ascites, hydrothorax; mural thrombus

Sac.—sacrificed
Tri.—tricuspid
Mur.—murmur
Ins.—insufficiency

interrupted sutures were placed in the right ventricle and the chamber opened between the sutures, after tension had been placed upon the sling ligatures about the superior and inferior venae cavae. Suction was applied. A leaflet of the pulmonic valve was caught with a dural hook and was excised with scissors. Some bleeding from the wound in the ventricle was encountered upon release of the sling ligatures after tying the sutures which had been previously placed. The bleeding was controlled by additional interrupted sutures. The pericardium was closed with interrupted sutures and the chest wound repaired.

On Jan. 19, 1939, the dog was up and walking. His condition was very satisfactory. Feb. 2, 1939, fluoroscopy showed definite cardiac enlargement predominantly of the right auricle, no edema of the feet, and no signs of venous congestion. On Nov. 13, 1940, the dog was sacrificed. See Table II for autopsy findings.

TABLE III

RESULTS OF TRICUSPID STENOSIS ALONE (7), COMBINED WITH MITRAL INSUFFICIENCY (1), AND OF PULMONIC INSUFFICIENCY (3) IN 11 DOGS

DOG	LESION	DURATION	DIED	SAC.	MUR.	PER.	GROSS PATHOLOGY	REMARKS
812	Tri. sten.	14 days	+		+		Ligature placed about a few chordae	More ins. than sten.
39	Tri. sten.	14 days	+		+	+	Ligature in medial cusp	Ligature cut out; thrombus
47	Tri. sten.	10 mo. 18 days		+	+		Slight deformity of valve	No cardiac decompensation
67	Tri. sten.	10 mo. 23 days		+	+		Opposite cusps tied together in middle of valve	No cardiac decompensation
522	Tri. sten.	Operation 5/7/41		Alive	+			No cardiac decompensation; appears healthy
523	Tri. sten.	Operation 5/14/41		Alive	+			No cardiac decompensation; appears healthy
612	Tri. sten.	Operation 7/18/41		Alive	+			No cardiac decompensation; appears healthy
11	Tri. sten. Mitral ins.			+	+		Good stenosis suture slit like ins. in aortic leaflet of mitral valve	More insufficiency than sten.; good lesion of mitral valve
80	Pul. ins.	18 days	+		+	+	One cusp almost removed	Lung abscess, empyema, purulent per., thrombosis, bronchopneumonia
258	Pul. ins.	11 mo.		+	+	+	One cusp removed	No cardiac decompensation
334	Pul. ins.	10 mo.		+	+	+	One cusp removed	Adhesions at the site of suture

Sac.—sacrificed Per.—pericarditis Sten.—stenosis
Mur.—murmurs Tri.—tricuspid Ins.—insufficiency

TABLE IV

RESULTS OF MITRAL INSUFFICIENCY (8), STENOSIS ALONE (6), AND IN COMBINATION WITH TRICUSPID STENOSIS IN 14 DOGS

DOG	LESION	DURATION	DIED	SAC.	MUR.	PER.	GROSS PATHOLOGY	REMARKS
487	Mitral ins.	10 min.	+				3 mm. opening in aortic leaflet of mitral valve	Died on table
A	Mitral ins.	10 min.	+				Portion of aortic leaflet of mitral valve excised	Died on table
152	Mitral ins.	18 hr.	+		+		Large portion of aortic leaflet of mitral valve excised	
91	Mitral ins.	Operation 12/11/41		Alive	+		Margin of leaflet and chordae excised	Good lesion
90	Mitral ins.	Operation 12/2/41		Alive	+		Chordae excised	Two attempts; lesion doubtful
613	Mitral ins.	20 hr.	+		+	+	Part of valve and chordae of aortic leaflet of mitral valve removed	Marked insufficiency; passive congestion of lungs
299	Mitral ins. Tri. sten.	21 days 12 mo.	+		+		T-shaped tear in mitral leaflet; 2 tricuspid leaflets removed	Thrombosis of coronary sinus, anasarca, asites, hydrothorax, mural thrombus
11	Mitral ins. Tri. sten.	3 mo. 18 days	+		+		Slitlike lesion in aortic leaflet; sten. of two leaflets	Good lesion of mitral valve; more insufficiency than stenosis
397	Mitral sten.	3 days	+		+		Valve and chordae of medial leaflet caught in ligature	Pneumothorax
384	Mitral sten.	2 days	+		+	+	Medial cusp and several chordae of opposite cusp tied together	Pneumonia right upper lobe, seropurulent exudate, both pleural cavities fibrinous, pleuritis
352	Mitral sten.	30 hr.	+		+		Ligature about chordae; one-half of valve margin included	Bilateral hemothorax, partial atelectasis of the right lower lobe
150	Mitral sten.	25 days	+		+	+	Suture uniting the two valves involving one-third the orifice	Thrombus of the left ventricle attached to valve and wall
239	Mitral sten.	18 hr.		+	+		Valve cusp partly torn; good bridge across valve opening	Nonsterile operation; dog was quite ill and would have died in a short time
254	Mitral sten.	60 hr.	+		+	+	Valve cusp partly torn, making a combined lesion	Died of pneumonia; vegetative endocarditis of mitral valve

Sac.—sacrificed Per.—pericarditis Ins.—insufficiency
Mur.—murmurs Tri.—tricuspid Sten.—stenosis

Dog 652.—On Oct. 4, 1938, a tricuspid insufficiency was formed by excising the leaflets of the tricuspid valve. The operation was made upon a normal healthy dog under intratracheal positive pressure ether anesthesia, following preoperative morphine and atropine. The incision was made in the fifth intercostal space on the right side; the superior and the inferior venae cavae were isolated and a sling ligature of black silk passed about the vessels, employing an aneurysm needle. The ozygos vein was tied off. The pericardium was opened longitudinally and a purse string of black linen placed in the right auricle, avoiding the appendage. Tension was put upon the sling ligatures and fifteen seconds allowed to pass so that the heart might empty itself of blood. The auricle was opened through the purse string. Suction was employed. The tricuspid leaflets were well visualized. The valve leaflets were grasped with tissue forceps and the medial cusp was cut away with scissors; a major portion of a second valve was removed in a similar fashion. The purse string was closed and the sling ligatures released. The pericardium was brought together with interrupted sutures. Then the chest wound was closed. Almost no cardiac embarrassment occurred; 50 to 60 c.c. of blood were lost. Two minutes were consumed during the operation upon the valves.

On Oct. 7, 1938, the condition was good. The animal was active. Oct. 25, 1938, fluoroscopy revealed definite right heart dilatation. There were no signs of decompensation. On Jan. 10, 1939, the dog was sacrificed (See Table II).

Dog 67.—On Nov. 22, 1938, a tricuspid stenosis was made by suturing the leaflet of one valve to another. Following a preoperative dose of morphine and atropine, an operation was made upon a normal healthy dog using intratracheal positive pressure ether anesthesia. The incision was made in the fifth intercostal space on the right side. The superior vena cava was freed and a sling ligature placed about the vessel, using an aneurysm needle. The azygos vein was isolated and a ligature placed about the vein. The vein was occluded by tension and the sling later released. The inferior vena cava was located and again using an aneurysm needle, a sling ligature was passed about the vessel. The pericardium was opened. A purse-string suture of black linen was placed in the wall of the right auricle. Tension was applied to the ligatures about the vena cava and fifteen seconds were allowed to pass for the heart to empty itself. The auricle was incised in the center of the purse string. After using the suction, a valvular lesion was created by suturing one leaflet to the other, using a small curved needle. After tying the purse-string suture the sling ligatures about the superior and inferior vena cava were released. The pericardium was closed with interrupted sutures and the chest wound repaired. Two and one-half minutes elapsed during the production of the lesion. Blood loss was about 60 c.c.

On Nov. 24, 1938, the dog was active, apparently recovered. Feb. 2, 1939, fluoroscopic examination revealed no significant change. On Oct. 10, 1939, the dog was sacrificed. See Table III and text for autopsy findings.

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TRAUMATIC HEMOTHORAX WITH SPECIAL REFERENCE TO CHRONIC PERSISTENT TYPES

H. G. Smithy, M.D.
Charleston, S. C.

HEMOTHORAX resulting from a closed wound of the chest wall represents the commonest variety of traumatic intrapleural bleeding seen in civil practice. The majority of such cases respond readily to the generally accepted measures of treatment, and recovery is usually prompt. A small group, however, fail to recover even in the absence of infection and show signs of persistent sterile hydrothorax with marked loss of vital capacity due to pleural fibrosis and consequent persistent pneumothorax. The problem presented by such cases is difficult to correct; it is this chronic recurrent or persistent variety with which the ensuing discussion is concerned.

MECHANISM OF THE INJURY

Intrapleural bleeding can be initiated by numerous traumatic agents. The commonest source, certainly in the deep south, is the negro social weapon, the "switch blade" knife. Penetrating wounds of the thoracic wall made by such a weapon are practically always simple puncture wounds and become immediately sealed off upon withdrawal of the instrument, thus forming a closed wound. The actual bleeding point responsible for the hemorrhage is generally situated in a laceration of the lung itself, although torn intercostal vessels, the internal mammary vessels, the great vascular system of the mediastinum, or the azygos vein may be at infrequent intervals the origin of the bleeding. Since the lung itself is responsible for hemorrhage in the majority of cases, pneumothorax is usually present so that the problem of management is concerned with hemopneumothorax in most instances. The degree of pulmonary collapse is governed by the amount of air escaping from the damaged lung parenchyma; the volume of air in turn depends upon the size of the rent in the alveoli and upon the involvement of one or more terminal bronchioles. In the latter instance pleural pressure may rise rapidly, whereupon a distressing train of events ensues. Where valvular flaps of torn lung result from the injury, allowing escape of air with each inspiration, tension pneumothorax is an almost invariable sequel.

The relationship of collapse of the lung to the amount of hemorrhage is interesting to note. Theoretically, as pleural pressure rises, incident to the pneumothorax, tamponade of the bleeding point by free air occurs.

From the Department of Surgery, Medical College of the State of South Carolina, and the Surgical Service of Roper Hospital, Charleston.
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However, in tension pneumothorax the victim grows apprehensive and excited, breathes deeply and rapidly, and coughs violently. Obviously, such activity causes increased motion of the torn lung, brisk flapping of the valvular opening, and, consequently, more hemorrhage. The inevitable result in such a situation, unless emergency measures are quickly instituted, is death due to shock. The latter results from compression of the mediastinum and the opposite lung, as well as from loss of blood into the pleural cavity. Fortunately, tension hemopneumothorax occurs infrequently and in those cases without tension bleeding often ceases as a result of the collapse alone.

MANAGEMENT OF THE ACUTE PHASE

It is generally agreed that certain preliminary measures are necessary in all thoracic injuries. These are concerned with (1) allaying excitement and apprehension, (2) treatment of shock, and (3) control of hemorrhage. Such features of treatment must precede any efforts at restoration of the cardiorespiratory apparatus to its normal status by surgical measures. In closed pleuropulmonary wounds, shock is commonly present and excitement and apprehension are almost invariable if hemopneumothorax has occurred. Beyond the preliminary stage of treatment, opinions begin to differ. Withdrawal of blood from the pleural cavity at varying periods of time after the injury is a common method of treatment, while restraint from thoracentesis and dependance upon spontaneous absorption of the effusion are practiced by others. Thoracentesis, with replacement of air or oxygen, performed two or three days after the injury is recommended by many[1-9] and seems to constitute the most popular management of the problem. Where massive hemorrhage with persisting shock, tension pneumothorax or pronounced dyspnea due to mediastinal displacement, and pulmonary compression have occurred, early thoracentesis or more extensive surgical measures are usually necessary.

An ultraconservative attitude is expressed by Elkin,[10] who states that "whenever possible the blood should be left in the chest."[11] In his experience, spontaneous absorption of the pleural blood occurs in 95 per cent of the cases. Taking an entirely different view, Schrire[12] recommends immediate exploratory thoracotomy in all types of pleuropulmonary injuries.

In summary, it appears that the treatment of acute or newly acquired traumatic hemothorax should follow a conservative trend. Delayed removal of pleural blood by simple aspiration is most generally employed except where uncommon circumstances demand surgical intervention. Results from such a routine are very good. Similarly, creditable results have been obtained by allowing the blood to be absorbed. Regardless of the measures employed, the fact remains that a small percentage of patients fails to recover completely and continues either to bleed further into the pleural cavity or to show persisting effusions.

Such cases are extremely difficult to cure and the problem presented by them has apparently received little attention in the literature.

The statistics in Table I were compiled from fifty cases of hemothorax and hemopneumothorax selected at random from the Roper Hospital files.

TABLE I

TYPE OF INJURY	NUMBER OF CASES	MORTALITY*	CHRONIC PERSISTENT HEMOTHORAX
Knife stabs	21	0	2
Bullet wounds	16	3	1
Fractured ribs	11	0	0
Ice pick stabs	2	1	0
Total	50	4 (8%)	3 (6%)

*Death occurred within forty-eight hours in each of these four patients; shock and hemorrhage were responsible in each.

No heart wounds are included in these figures.

No deaths occurred in the forty-six patients in Table II. Open thoracotomy was performed in three cases because of infected hemothorax and in one case because of chronic persistent hemothorax (Case 1). Exploratory thoracotomy with snug closure of the wound was performed in one patient because of persistent hemothorax (Case 2).

TABLE II

TREATMENT

CONSERVATIVE	NUMBER
Bed rest alone (spontaneous absorption)	20
Thoracentesis alone	8
Thoracentesis with air replacement	13
RADICAL	
Open thoracotomy	4
Exploratory thoracotomy with closure	1
Total	46

THE CHRONIC OR PERSISTENT PHASE

Since a high percentage of cures is obtained by both aspiration of pleural blood and the spontaneous absorption method of Elkin, it appears that little or nothing can be done to prevent that small but definite group of patients from entering the chronic phase. Apparently an occasional case of traumatic hemopneumothorax or hemothorax alone will progress to the stage of loculation and recurrent effusions regardless of the variety of treatment employed in the acute stage. It is interesting to speculate upon the mechanism of this chronicity. Those who recommend evacuation of blood shortly after the injury do so because of certain undesirable effects of the blood on the pleura. Edwards and Davies[8] have studied the response of the pleura to blood by thoracoscopy. Their findings indicate that the pleural reaction is inflammatory and that the effects of the blood are distinctly irritative. Acute hyperemia, edema, fibrin deposition, trabeculation, and finally organization of fibrin

with fibrous attenuation of the entire lung have been observed by these authors. Thompson[13] states that all hemothorax patients develop serous pleural effusions as a result of the irritating effects of blood. This feature is readily demonstrated by determining the hemoglobin content of pleural blood at intervals. It will be found high immediately after the trauma (50 to 70 per cent) but rapidly declines as serous pleural fluid accumulates and dilutes the blood. If evacuation of blood is incomplete or absorption is inadequate, loculation occurs and the serous fluid collects in numerous pockets. After several weeks, organized adhesions appear throughout the loculated field, the visceral pleura becomes thickened, and pulmonary collapse is then maintained. Proceeding still further, cholesterol crystals are deposited in the encysted fluid[6] and eventually calcification may occur. Hugonot,[14] commenting on three patients with persistent hydrothorax whose histories dated back to World War I, states that extensive calcium deposits were noted throughout the affected pleural cavity when x-ray studies were made in 1932 and 1938. He reports a fourth patient injured in 1929 (hemopneumothorax), and showing pleural calcification in 1940. Two of these patients had an atrophy of one-half of the thorax and two had atrophy plus compensatory scoliosis. All were debilitated. Such, apparently, is the likely end result in that small group of individuals who fail to get rid of their pleural blood by either spontaneous absorption or surgical evacuation.

TREATMENT OF THE CHRONIC PHASE

Comments on the management of a posttraumatic persistent hydrothorax should be introduced by the statement that results that I have obtained thus far are discouraging and neither a formal routine nor even a satisfactory set of procedure of approach to the problem has been established. However, the cases reported herein serve as a basis for further study and are informative in spite of a low incidence of cure.

Treatment in the chronic phase, as in the acute, divides itself into conservative and radical. Recalling the rather striking experiences of Hugonot,[14] one is impressed by the necessity of removing, by one means or another, the loculated fluid collections of the pleura. However, calcification, pleural fibrosis, and hemiatrophy of the thorax are late sequelae and hence the advisability of procrastination becomes a matter for considerable thought. No doubt, some cases of persisting hydrothorax will eventually clear up spontaneously. The pressing point of judgment lies in deciding when one must abandon conservative waiting and resort to operative treatment. It appears logical to assume that where loculation is well defined and radiographic evidence of pleural thickening is marked, spontaneous cure will be a most remote possibility. Under such circumstances, removal of the offending agents from the pleural cavity offers the only means of cure. Following the conservative trend, thoracentesis and closed intercostal catheter drainage may be

tried. Since the object of treatment is to restore the cardiopulmonary status of the patient to normal, it is obvious that neither aspiration nor intercostal drainage can accomplish a re-expansion of the lung through its fibrous pleural coat. Furthermore, neither method can remove the organized network of pleural adhesions. Thus, one must depend largely upon x-ray studies as a guide to determining the time for radical treatment.

Thus far, no mention has been made of infection. Where infected hemothorax occurs, the case must be treated by open rib resection at the earliest moment compatible with tolerating an open pneumothorax. In the first case reported, open thoracotomy was performed despite the fact that no evidence of infection was noted at operation. The pleural cavity was cleaned of its contents and the operative wound left open. A fulminating empyema developed which almost cost the patient his life. Thus was grapically illustrated the fact that where exploration of the chest is contemplated, the wound should never be left open unless the hematoma is infected. Even the use of a catheter for drainage, with the wound sutured snugly around it, is to be condemned, so great is the susceptibility of the pleura to infection in the presence of organizing thrombi.

When radical operative treatment has been decided upon, the one remaining procedure is exploratory thoracotomy through a large incision. Complete evacuation of clots, fibrin, fluid, and friable bands having been accomplished, the wound must be closed firmly. This procedure is mentioned by Butler[15] as being of value in aborting empyema from low-grade infected hemothorax. The local implantation of one of the sulfonamide crystals seems to have its virtues. Where the visceral pleura is found to be unusually thick, an attempt at stripping should be made or the "cross-hatching" procedure of Ransohoff carried out. Immediately following firm closure of the wound, aspiration of air should be done until a negative pressure is reached, thereby aiding further in expanding the lung. Effusion is common following exploration of the thorax and may necessitate frequent aspirations at intervals.

CASE REPORTS

The following three patients are selected as typical of chronic persistent hemothorax. They are reported because each was treated in a different manner and the various methods used entail all the known conservative and radical varieties of treatment.

CASE 1.—A 35-year-old white mechanic entered the hospital Feb. 23, 1942, a few minutes following infliction of a stab wound in the left posterior axillary line at the sixth interspace. He was in profound shock on arrival at the emergency room but responded satisfactorily to a plasma infusion. During the first forty-eight hours following admission, the patient developed signs of hemopneumothorax, and x-ray con-

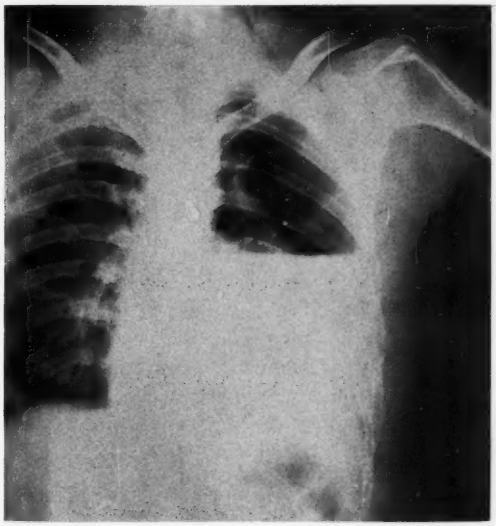

Fig. 1.—Admission findings in Case 1. Note the well-defined hemopneumothorax.

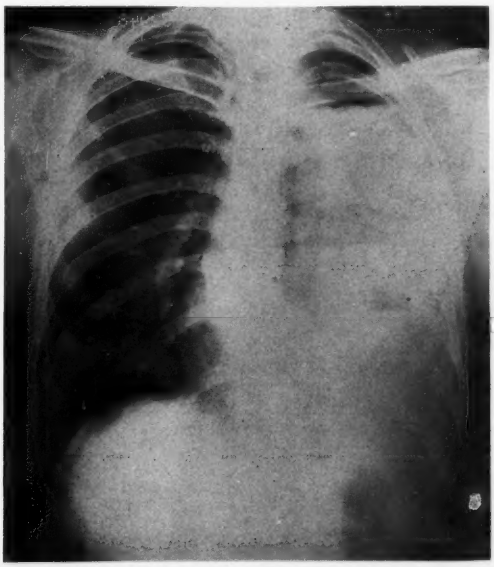

Fig. 2.—Loculation and encapsulated fluid pockets of patient in Case 1, eight weeks after injury.

firmed the diagnosis (Fig. 1). Three days after admission, thoracentesis was done and 250 c.c. of blood removed from the left pleural cavity. Replacement of 125 c.c. of air was made. Reaccumulation of fluid was prompt and one week after admission 1200 c.c. of serosanguineous fluid were withdrawn by aspiration. Culture of a specimen of this was sterile. The patient was discharged fourteen days after the injury with radiographic evidence of much reduction in the amount of pleural fluid and some improvement in the degree of pulmonary collapse.

Outside the hospital, the patient suffered from pain in his chest, dyspnea, and persistent elevation of temperature. He was readmitted April 22, 1942, at which time an x-ray film showed multiple fluid levels in the left pleural cavity (Fig. 2). Several attempts were made to aspirate the fluid but none was productive of more than a few cubic centimeters of serum and degenerating blood. Two days after admission, a mushroom catheter was placed in the pleural cavity by means of a trocar and cannula through the eighth left interspace, and closed drainage under water was established. Great improvement in dyspnea and decrease in temperature followed this procedure but there was no significant change in the x-ray findings five days later.

On May 5, 1942, an open thoracotomy was done by subperiosteal resection of four inches of the left eighth rib in posterior and midaxillary lines. Five hundred cubic centimeters of brownish odorless fluid were evacuated. There were many pockets of fluid noted and numerous organizing thrombi were present. Both pleural surfaces were covered by deposits of thick shaggy fibrin. The total capacity of the hemothorax cavity was estimated at 1000 c.c. The wound was packed open. Subsequent course of the patient was distressing and alarming. An overwhelming infection of the entire left pleural cavity developed and the patient remained in a critical condition for two weeks. Such was the extent of the empyema that, when recovery finally occurred, the patient was left with a tremendous cavity which necessitated a later thoracoplasty for closure.

Case 2.—A 40-year-old Negro laborer sustained a stab wound of the chest near the left nipple on May 23, 1942. He was admitted to the hospital shortly after the injury in a severe degree of shock which persisted for several hours in spite of blood transfusions and the usual shock therapy. A left hemopneumothorax was present on admission (Fig. 3). Treatment of this patient was similar to that in Case 1, an attempt being made to empty the pleural cavity by several interval thoracenteses. Repeated effusions of serous fluid occurred following aspirations but the patient was discharged from the hospital afebrile and symptom-free on June 9, 1942, seventeen days after admission. Cultures made at each aspiration were persistently sterile. A small amount of fluid remained in the left pleural cavity and a moderate degree of pneumothorax was also present at the time of discharge.

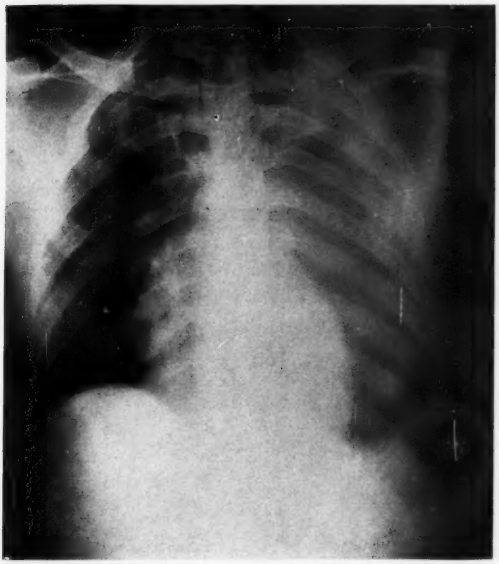

Fig. 3.—Admission findings in Case 2 showing opacity due to left hemothorax.

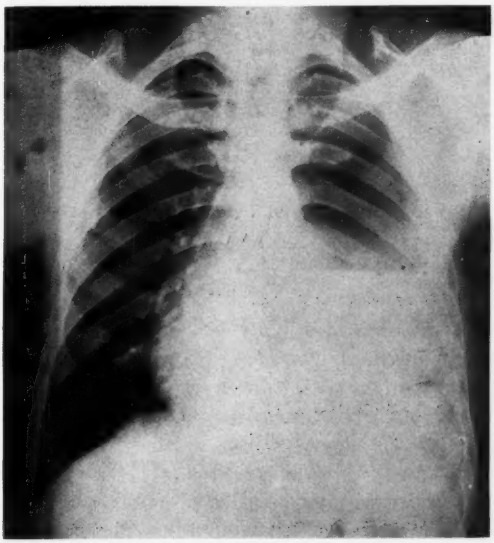

Fig. 4 (Case 2).—Patient four weeks after injury, showing persistent hemopneumothorax.

Follow-up visits in the outpatient department showed this patient to be suffering from constant deep chest pain, elevation of temperature, and dyspnea. Repeated efforts at thoracentesis were unsuccessful and the patient was readmitted to the hospital June 23, 1942. Radiographic studies at this time revealed persistent hemopneumothorax (Fig. 4). One thoracentesis shortly after admission was productive of 600 c.c. of reddish-yellow fluid which showed no growth on culture. Rapid reaccumulation of fluid took place so it was felt that removal of the pleural fibrin and clots was necessary. Accordingly, an exploratory thoracotomy was done under ethylene gas anesthesia July 7, 1942, some six weeks after the injury, the pleura being entered by subperiosteal resection of seven inches of the left sixth rib. There was found a tremendous amount of chicken fat clot and organizing thrombi which

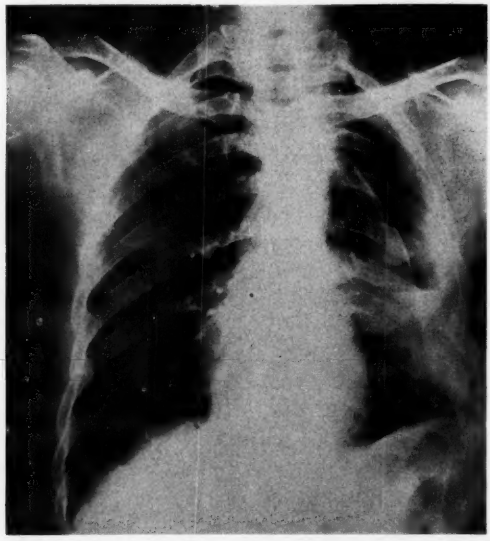

Fig. 5 (Case 2).—Patient six weeks after exploratory thoracotomy with closure. Pleural fibrosis hinders expansion of left lung.

divided the pleural space into many loculated pockets, some of which contained clear yellow fluid. The lung was almost completely collapsed and both parietal and visceral pleura were somewhat thickened and covered with shaggy fibrin. Complete removal of all fibrin and clots was effected, leaving the pleural surfaces clean. The wound was closed snugly in layers with interrupted sutures of black silk. Aspiration of air was done after closure until a negative pressure was reached.

Recovery of the patient following operation was punctuated by numerous aspirations of serosanguineous fluid incident to the trauma of operation. He was discharged symptom-free July 30, 1942, twenty-three days after operation. However, radiographs made at the time of dis-

charge showed persistence of the pulmonary collapse but great reduction in the amount of pleural fluid.

The course after discharge from the hospital was one of steady improvement. The patient returned to work as a hospital orderly and on Aug. 24, 1942 an x-ray examination revealed his effusion to be definitely subsiding, although a considerable degree of pneumothorax remained and the extent of visceral pleural fibrosis was striking (Fig. 5). Despite these findings, the patient continued symptom-free. On Dec. 22, 1942, further serial roentgenographs were taken. At this time, the affected lung was seen to be completely expanded and only an insignificant amount of fluid remained (Fig. 6). Symptomatically, this patient is in excellent circumstances and has returned to a regime of unrestricted activities, some six months having elapsed since operation.

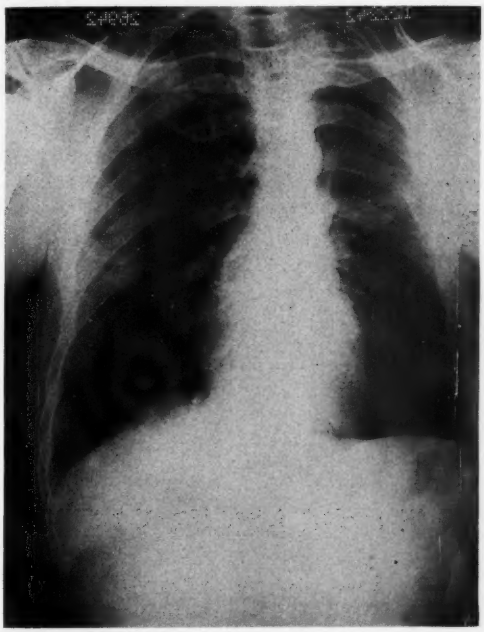

Fig. 6 (Case 2).—Patient six months after operation, showing re-expansion of left lung. Note the very slight fluid level in the left base.

CASE 3.—A 40-year-old white woman received a pistol-shot wound Dec. 30, 1941, the bullet entering the left second intercostal space 2 cm. from the sternum. Two hours later she reached the hospital and was admitted in profound shock which responded favorably to prompt treatment. A wound of exit was found just below the angle of the right scapula. Physical signs of right hemothorax were definite; there was no evidence of injury to the heart. A radiogram on admission confirmed the impression of hemothorax (Fig. 7).

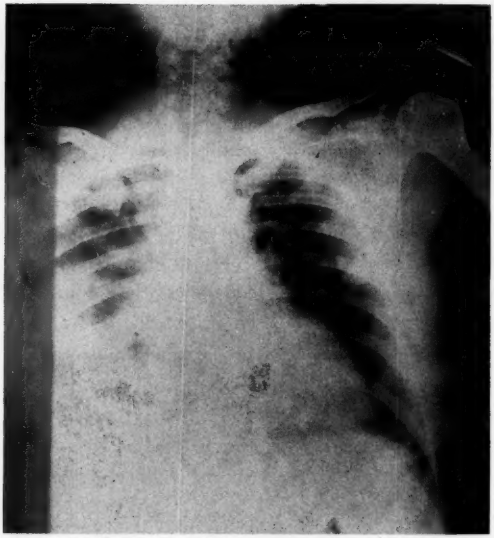

Fig. 7.—Admission findings of right hemothorax in Case 3.

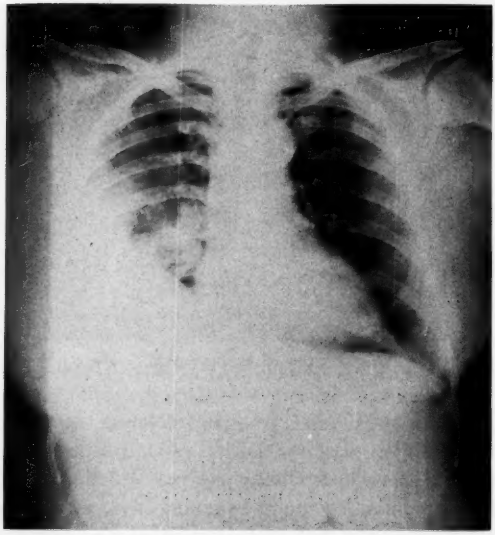

Fig. 8.—Persistence of hemothorax in Case 3, four weeks after injury.

On the fourth day after infliction of the injury, thoracentesis yielded 300 c.c. of bloody fluid; 200 c.c. of air were replaced. Thereafter, every attempt at aspiration of pleural blood met with failure. The patient improved steadily despite a constant febrile reaction and was discharged symptom-free Jan. 30, 1942, exactly one month after her injury. A chest plate at the time of discharge showed persistence of the hemothorax (Fig. 8).

This patient was not seen again until Sept. 9, 1942. On that date, complete history and physical examination revealed several interesting facts. Exertional dyspnea was present but not pronounced. Frequent stabbing pains in the right lower thorax occurred. There was present a pronounced splinting of the right hemithorax on forced inhalation, while auscultation and percussion showed high fixation of the right dome of the diaphragm and no basilar excursion of the right lung on inspiration. X-ray corroborated these findings (Fig. 9).

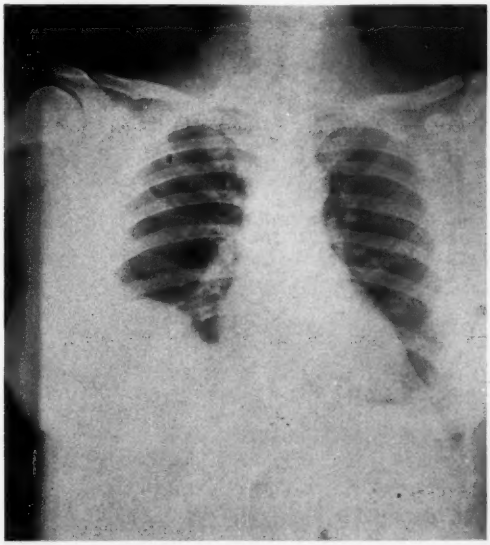

Fig. 9 (Case 3).—Patient after nine months of conservative treatment. Fixation of diaphragm and limitation of excursion of right lung are marked.

SUMMARY AND CONCLUSIONS

1. Conservative treatment is the preferable form of management in acute traumatic hemothorax. Good results are obtained from both aspiration and the spontaneous absorption method.

2. Regardless of the method of conservative treatment employed in newly acquired traumatic hemothorax, a small percentage of cases will enter the chronic persistent phase and will show pleural thickening, fluid loculation, and persistent pneumothorax.

3. Evidence is available to indicate that blood exerts an unfavorable effect on the pleura, creating an inflammatory reaction which may lead to the changes noted in chronic persistent hemothorax. Extensive pleural calcification with hemiatrophy of the thorax has been noted as a late sequel in some persistent cases.

4. In chronic persistent cases which are uninfected, exploratory thoracotomy with removal of all fibrin, clots, and fluid is recommended; the wound must be snugly closed without drainage.

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BRONCHIAL ADENOMA WITH METASTASIS TO THE LIVER

WILLIAM M. ANDERSON, M.D.
ST. LOUIS, MO.

DURING the past ten years there have been many reports in the literature concerning bronchial adenoma. There has, however, been no general agreement among either pathologists or clinicians as to just what type of tumor the bronchial adenoma represents. Some workers have considered it to be a malignant tumor and capable of metastasizing,[6, 14] while others have thought it benign.[8, 10] Up until 1937, there had been no report of a case with metastases, but since that time there have been several examples described in the literature. In all of these, however, the metastasis either was to an adjacent lymph node or it was a microscopic metastasis in a vertebra or in the liver. The literature thus contains reports of only two instances in which distant metastases occurred.[14] The case which is to be reported here presents definite evidence of such a metastasis and, therefore, provides further support for the belief that bronchial adenomas should be regarded as potentially malignant tumors.

REPORT OF CASE

History (Barnes Hospital No. 98541).—A 40-year-old white man entered Barnes Hospital, June 30, 1942, on the Medical Service of Dr. Harry Alexander, with the chief complaint of attacks of "asthma" for three or four years. His past history was notable in that he had pneumonia in 1934, 1937, and 1940, and had felt rather weak for several months following each attack. During the present illness there had been many sudden attacks of dyspnea, most of which were relieved by adrenalin. On Jan. 6, 1942, however, he had a sudden and severe episode of dyspnea which was not relieved by adrenalin, and during the ensuing six months he suffered numerous similar attacks. He had not been able to work since January, 1942. The dyspnea was characterized by easy inspiration and labored expiration, and the patient himself could hear bubbling noises in his chest. There had never been any cough or hemoptysis until six months prior to his admission to Barnes Hospital. From then on, however, his cough became progressively worse and productive of a thick mucoid sputum. During two attacks he coughed up a small amount of blood. He thought that during the preceding six months he had lost about twenty pounds, also, he had become progressively weaker.

From the Department of Pathology, Washington University School of Medicine, St. Louis.
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Physical Examination.—The temperature was 37.4° C.; pulse rate, 85; respiratory rate, 20; and blood pressure, 115/75. The patient was well developed yet rather poorly nourished. He did not appear acutely ill. Physical abnormalities were limited to the thorax. Expirations were labored and the right side of the thorax appeared to remain expanded slightly longer than the left. There were no areas of dullness, but instead, the percussion note over the entire chest was hyperresonant. Auscultation revealed numerous râles throughout both lung fields. Cardiac dullness was not easily made out, and the cardiac sounds were distant. The rate and rhythm of the heart were normal. A laryngoscopic examination was made and the examiner stated that he believed he could see a smooth, round red mass at about the fourth tracheal ring.

Laboratory Data.—The erythrocyte count was 5,650,000 and the hemoglobin content was 105 per cent. The leucocyte count was 6,400 with 64 per cent polymorphonuclear leucocytes, 22 per cent lymphocytes, 11 per cent monocytes, and 3 per cent eosinophiles. The Kahn test was negative; the urine was normal; the sputum contained no acid-fact bacilli.

X-ray Examination.—A posteroanterior view of the chest revealed some enlargement of both hilar shadows and rather coarse lung markings, but clear parenchyma. A laminogram showed a soft tissue mass at the right hilum, rather circular in outline and about 3.5 cm. in diameter. In a lateral view this mass appeared to be in the posterior mediastinum.

Course.—Bronchoscopy was scheduled for July 3, 1942, but on the preceding night at midnight a sudden attack of dyspnea developed. Treatment, consisting of adrenalin and an oxygen tent, was unsuccessful and the patient died forty-five minutes later.

Autopsy (Washington University, Department of Pathology, No. 9890).—The autopsy was performed six hours post mortem. Externally the body showed no pathologic changes. Over the right lung there were numerous moderately firm, fibrous, pleural adhesions. There were no adhesions over the left lung and there was no excess fluid in either pleural cavity. The lungs weighed 1200 Gm. and were somewhat distended. There were numerous blebs over the surfaces of the lungs, and the parenchyma of all lobes was doughy in consistency. There were no areas of increased firmness. There was a calcified nodule, 2 mm. in diameter, in the periphery of the lower lobe of each lung. Just beneath the pleura of the lower lobe of the left lung there were numerous grayish white, firm nodules, each about 1 mm. in diameter. Cut section of the lungs revealed no areas of consolidation and no bronchiectasis. Arising in the right main bronchus just below the bifurcation of the trachea there was a large (6 cm. in greatest diameter) firm, rounded, pink mass projecting into the lumen of the bronchus (Fig. 1). This new growth of tissue extended to a point just above the carina where it

almost completely occluded the lumen of the trachea. The base of the tumor was broad and extended into the bronchial wall but not beyond it. Cut section of this mass revealed a uniformly grayish-white tissue with a few small areas of hemorrhage near the center. The lymph nodes of the tracheal, right and left bronchial, and right and left bronchopulmonary groups were slightly enlarged and firm. The liver weighed 1900 Gm. and was dark in color; cut section revealed the normal lobular markings. Near the inferior border of the left lobe of the liver there

Fig. 1.—Bronchial adenoma arising in the right main bronchus.

was a slightly elevated, round, firm, grayish-white nodule 2 cm. in diameter. The heart was slightly enlarged, weighing 380 Gm. There were small foci of fibrosis in the myocardium and there was slight arteriosclerosis of the coronary arteries. Other than the minor abnormalities listed in the final diagnosis below, there were no other pathologic changes noted in the spleen, kidneys, pancreas, adrenals, reproductive organs, and bladder. The brain was not examined.

Microscopic Examination.—A section of the tumor of the bronchus showed it to be composed of a number of lobules separated from each other by fibrous tissue septa (Fig. 2). Each lobule was made up of

many alveolar-like spaces containing varying amounts of a granular, faintly acidophilic material. The walls of the "alveoli" were composed of medium sized cells, which were irregular in outline and had a moderate amount of clear cytoplasm (Fig. 3). The nuclei of these cells were round or oval shaped, moderately hyperchromatic, and contained reticulated chromatin material. Occasional nucleoli were present. There were no mitoses. There was some variation in the size and shape of these cells, but this was not marked. There was a small amount of fibrous tissue stroma. Some areas of the tumor had large vascular spaces which were engorged with blood. Normal ciliated columnar bronchial epithelium just above the tumor could be seen to undergo a

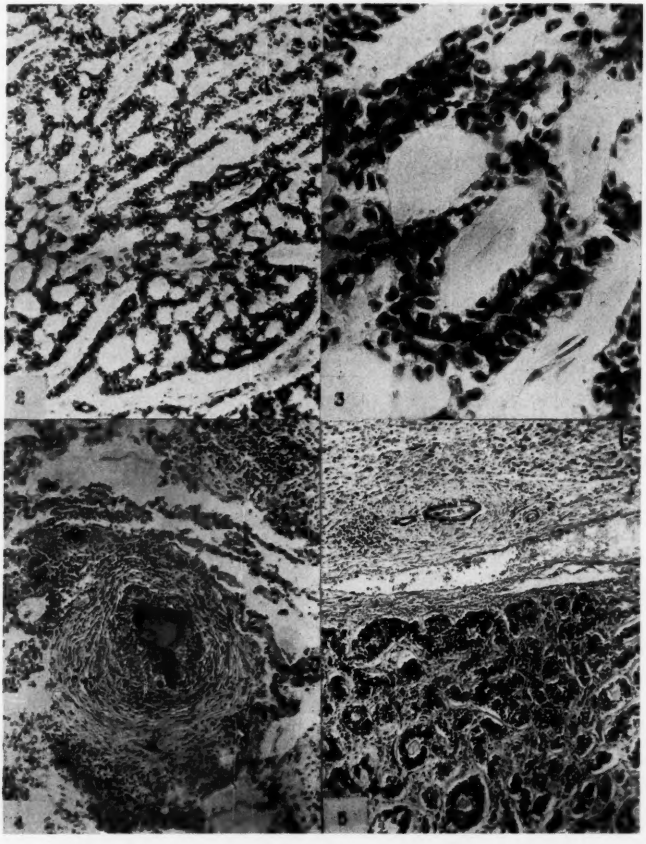

Fig. 2.—Photomicrograph of primary tumor showing alveolar arrangement (×84).

Fig. 3.—Higher magnification of Fig. 2 (×353).

Fig. 4.—Photomicrograph showing nodule of sarcoidosis in lower lobe of left lung (×68).

Fig. 5.—Photomicrograph showing liver tissue above and metastasis of bronchial adenoma below (×68).

definite transition as it began to cover the tumor. Covering the tumor there was stratified squamous epithelium, beneath which, in places, was a thick layer of fibrous tissue. The cells of the tumor did not invade the bronchial cartilages, but did extend between them to involve the entire thickness of the bronchial wall. Neoplastic cells were also present in several perivascular and perineurial lymphatics near the periphery of the tumor. Sixteen sections taken from various parts of the mass did not reveal the presence of abnormal bone, cartilage, or smooth muscle. Special stains failed to demonstrate argentaffin granules in the tumor cells.

A section of one of the lymph nodes adjacent to the tumor showed the architecture to be greatly distorted by the presence of many closely packed nodules, some of which contained Langhans' giant cells in the center. Surrounding the giant cells were epithelioid cells and then fibrous tissue arranged in circular fashion. There was no caseation. The node contained no cells similar to those in the bronchial tumor.

A section of the lower lobe of the left lung in the area where small nodules were seen grossly beneath the pleura showed each nodule to be made up of a Langhans' giant cell at the center, surrounded by epithelioid cells and fibrous tissue (Fig. 4). None of these showed any caseation. Except for moderate emphysema the remaining pulmonary tissue was normal. Acid-fast stains did not reveal the presence of acid-fast organisms in the lymph node or subpleural nodules. A diagnosis of sarcoid of the lung and lymph node was made.

A section of the nodule in the liver showed it to be composed of tissue identical with that in the tumor of the bronchus (Fig. 5). The acidophilic granular substance in the "alveoli" of the nodule in the liver was slightly larger in amount than that in the "alveoli" of the primary tumor. The "alveolar" walls in the former were slightly thicker and more cellular than in the latter. Also, the fibrous tissue stroma of the metastatic nodule was a little more abundant than in the bronchial tumor. There was no capsule around the nodule in the liver, and hepatic cells were seen at various points just inside the tumor. The surrounding liver cells were flattened and atrophic. The sinusoids in the vicinity were dilated.

Sections of myocardium, spleen, pancreas, and kidney revealed no pathologic changes other than those listed below.

The complete anatomic diagnosis was: malignant adenoma of the right bronchus with stenosis of the right bronchus and trachea; metastasis in the liver; emphysema of the lungs; sarcoidosis involving the tracheobronchial and bronchopulmonary lymph nodes and the subpleural lymphatics of the lower lobe of the left lung; fenestrations of the cusps of the aortic and pulmonary valves; adhesions at a commissure of the aortic valve; fibrous thickening of the leaflets of the tricuspid and mitral valves; arteriosclerosis of the aorta and coronary arteries; sclero-

sis of the anterior leaflet of the mitral valve; focal fibrosis of the myocardium; fibrous pleural adhesions; cholesterolosis of the gall bladder; calcified nodules in the lower lobes of the lungs; focal hyperplasia of the renal tubules.

DISCUSSION

No attempt will be made here to review the entire literature on this subject, as this has been adequately done by Foster-Carter[1] in 1941. However, it will be helpful to discuss this case in relation to the prevailing ideas concerning bronchial adenoma. This will be done under the two headings Origin and Malignancy.

Origin—Mucous glands: There seems to be little doubt that there is some relation between bronchial adenoma and the mucous glands of the bronchial tree. These tumors have always arisen in the larger bronchi and since the smaller bronchi have no mucous glands, this is a point in favor of the origin of bronchial adenoma from the mucous glands.[2] Kramer[3] goes further in stating that such neoplasms arise from the ducts of the mucous glands, and he gives as his reasons the following evidence: (1) The cells resemble each other; (2) he has seen the mucous gland ducts proliferating in the region of such a tumor; and (3) he cites the case of Horn in which the acini of a bronchial adenoma were lined by ciliated epithelium. Kramer's view as to the origin of these tumors is probably the most widely held at present. The fact that the tumor which I am presenting was composed almost entirely of glandlike tissue containing mucoid material is strong evidence that it originated from the bronchial mucous glands. In this instance, however, nothing has been found which would support the theory that the tumor arose from the ducts of the mucous glands.

Relation to carcinoid tumors: Some pathologists have noted the similarity of histologic appearances of the bronchial adenoma and the carcinoid of the appendix. However, the only instance in which argentaffin granules have been demonstrated in the cells of bronchial adenoma is reported by Hamperl,[8] and even he was not certain that he had actually shown silver-positive granules to be present. Others have attempted to prove the relation to carcinoid but have never been successful.[4] In the case presented here special stains failed to demonstrate argentaffin granules in the cells of the bronchial tumor. It must be concluded then that any relation of bronchial adenoma to carcinoid is far from proved.

Relation to mixed tumors of the salivary glands: Brock[5] and Womack and Graham[6] have postulated that bronchial adenomas originate from two germ layers as do the mixed tumors of the salivary glands. Brock based his opinion mainly upon the apparent origin of bronchial adenoma from mucous glands and the great similarity which it shows to the parotid tumors in clinical course. Womack and Graham have studied the subject much more extensively and have even used the name "mixed

tumor'' to designate bronchial adenoma. They believe these tumors to be composed of both mesodermal and entodermal elements in varying proportions. At the mesodermal extreme they place chondromas, osteomas, and fibromas, while at the entodermal extreme are the tumors which have been called bronchial adenomas. Between these two extremes there are tumors which have a more obviously mixed constitution. To support this idea they report several cases of tumors similar to bronchial adenomas but containing both mesodermal and entodermal elements, the former being in the form of bone, cartilage, and smooth muscle, while the latter is represented by glandlike structures. The only other reports in the literature of the presence of mesodermal components in bronchial tumors are the two cases of Brunn and Goldman[7] and two of Adams, Steiner, and Bloch.[14] The tumor in each of these four instances contained spicules of bone. In the case which I am reporting a careful microscopic search through sixteen sections taken from various parts of the bronchial tumor failed to reveal the presence of any mesodermal elements. The findings in this instance, therefore, do not support the contention of Womack and Graham that these are mixed tumors. It must be admitted, however, that the tumor reported in this paper might represent the extreme entodermal end of their scale and hence contain no mesodermal elements. Since there was a metastasis, this conception of its status would fit in with Womack and Graham's idea that the tumors in which entodermal elements predominate are more malignant than the others. However, until more cases of bronchial adenomas continuing mesodermal elements are reported, the evidence for their mixed nature must be considered inconclusive.

Relation to cylindromas: Most pathologists use the term ''cylindroma'' to refer to a special type of basal-cell carcinoma occurring especially on the scalp,[16] and sometimes spoken of as a ''turban tumor.'' According to Ewing,[15] however, ''cylindroma'' may be used to designate any tumor ''in which the stroma appears in the form of elongated, twisted, thickened cords of hyaline substance.'' Hamperl[8] and Kramer and Som[9] have described tumors of the trachea and bronchi which they call cylindromas. Kramer and Som distinguish between these tumors and the ordinary bronchial adenoma, the former being characterized by alveolar-like spaces containing hardened mucoid material in the form of cylinders, while the latter are composed of smaller nests of cells forming acinar structures. They believe cylindroma of the bronchus to be fundamentally the same tumor as the cylindroma which is related to basal cell carcinoma. Hamperl uses the term cylindroma to designate the same tumors which have been called bronchial adenomas, because in many of them there is a cylindromatous type of stroma. Although the tumor in the case I am reporting is similar histologically to the cylindromas described by Kramer and Som, it seems unlikely that it is essentially different from all of the others which have been called

bronchial adenoma. The term, cylindroma, as used in designating bronchial adenoma should, therefore, be regarded as a descriptive name only and not implying any fundamental relation to the cylindromatous type of basal cell carcinoma.

Malignancy.—The question as to whether or not the bronchial adenomas are malignant is one which has not been answered satisfactorily; yet it is vitally important to know the answer to this question because the basic principles of treatment will depend upon whether or not it is malignant. Everyone is agreed that if left untreated the tumor will grow slowly and produce obstruction and infection which will eventually kill the patient;[10] but most pathologists have considered the tumor to be essentially benign from a pathologic point of view.

Malignant changes in the primary tumor: Occasionally there have been references made in the literature to a malignant transformation of bronchial adenoma.[10, 11] Some of these have been based on the presence of tumor tissue on both sides of the bronchial cartilages, but Wessler and Rabin[10] state that this is no proof of malignancy because normally there are mucous glands on both sides of the cartilages. Reisner[11] considered the presence of occasional mitoses, hyperchromatic nuclei, and some variation in the size and shape of the cells to be evidence of malignant change. In the case which I am reporting there was involvement of both sides of the bronchial cartilages, and the nuclei were moderately hyperchromatic, but there were no mitoses. Except for the presence of neoplastic cells in several small lymph channels the general histologic appearance of the primary bronchial mass alone was considered benign by all who examined it. It was mainly because of the presence of a distant metastasis that the diagnosis of "malignant adenoma" was made.

Metastasis: The first report of a metastasis of a bronchial adenoma was given in a paper by Zamora and Schuster,[12] in 1937, when they stated that W. E. Barnard in a personal communication to them reported the occurrence of a metastasis. They did not, however, give any details concerning this. Mallory,[13] at the Massachusetts General Hospital, observed only one example of a metastasis of a bronchial adenoma to a regional lymph node. Womack and Graham[6] also mention a local metastasis. They have considered these tumors as being potentially malignant and have, therefore, treated their patients with lobectomy and sometimes pneumonectomy. The only instance in which distant metastases were described and details given was by Adams, Steiner, and Bloch,[14] in 1942. They report three cases in one of which a 62-year-old woman had a typical adenoma in the bronchus to the lower lobe of the right lung and at autopsy was found to have a microscopic metastasis in the body of a vertebra. This was observed incidentally in a routine section. A second patient was a 47-year-old man who had a large (6 cm. in diameter) bronchial adenoma in the right lower lobe bronchus and a microscopic metastasis in one tracheobronchial lymph

node. The primary tumor in this patient also contained two small "calcified bodies." A third case was that of a 36-year-old woman who had a large (7 by 10 by 8 cm.) bronchial adenoma in the right main bronchus. In this instance, also, the tumor in the bronchus contained spicules of bone and there was a microscopic metastasis in a lymph node adjacent to the tumor; and in one portal zone of a liver lobule there were seen two small groups of cells which were thought to be metastases.

There is no explanation for the occurrence of the distant metastasis in the case which I am reporting other than to assume that these tumors are potentially malignant and do occasionally metastasize. The only evidence of malignancy in the primary tumor in this case was invasion of several lymph vessels. This has also been noted by Womack and Graham in their cases. Although this patient's illness apparently went back only two or three years, it is a fact that he had attacks of "pneumonia" in 1934, 1937, and 1940. At autopsy the presence of numerous fibrous pleural adhesions over the right lung and not over the left lung are further evidence of some previous pulmonary infection, apparently localized to the right side; and it was in the right main bronchus that the bronchial adenoma was located. The size of the tumor (6 cm. in greatest diameter) is further evidence that it must have been there for a number of years. After examining the cases in the literature which have reported both the size of the bronchial adenoma and the duration of symptoms it was noted that whenever the greatest diameter of the tumor exceeded 5 cm., the duration of symptoms was over eight years. It can be safely assumed, therefore, that this tumor ran a course of at least eight years and probably more. It is possible that the presence of the tumor in this patient over such a long period of time may have been a contributing factor in the occurrence of the metastasis. This phenomenon has occurred in cases of other slightly malignant tumors such as basal-cell carcinomas and mixed tumors of the parotid gland.[17, 18] The presence of another disease, Boeck's sarcoid, in the same patient must be considered a purely incidental finding and one which bears no relation whatsoever to the bronchial adenoma.

SUMMARY AND CONCLUSIONS

A case is here reported of a tumor of the bronchus which alone would be considered a benign bronchial adenoma, but which was diagnosed as malignant upon the finding of a metastasis in the liver. The various ideas concerning the origin of bronchial adenoma are discussed and it is concluded that these tumors may be mixed tumors as postulated by Womack and Graham, but that sufficient evidence for this hypothesis is lacking, at present. The case presented here provides, however, definite proof that metastases can and do occur and it suggests, therefore, that the principles of treatment for bronchial adenoma should be the same as for other malignant tumors.

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THORACOSCOPY IN PULMONARY TUBERCULOSIS

JOSEPH GOORWITCH, M.D.
SAN FERNANDO, CALIF.

INTRODUCTORY STATEMENT

THIS study was stimulated by a remark from a fellow physician in the course of a staff conference at the Olive View Sanatorium. The gist of the remark was that inasmuch as thoracoscopy was essentially a surgical procedure, it was not indicated in therapeutic pneumothorax in which pneumonolysis was apparently not feasible judging by the appearance of the adhesions in the roentgenograms.

As pointed out by many workers[1-5, 7, 8, 10, 12-14] and recently stressed by John C. Jones,[6] the only reliable method of determining feasibility of closed pneumonolysis is inspection of the adhesions in the course of thoracoscopy; roentgenography, though very helpful, is not without its limitations. Employing this concept as a starting point for further reasoning, it would seem inadequate therapy not to give the patient the benefit of thoracoscopy in instances in which pneumonolysis does not seem to be entirely out of the question by reason of diffuse adherence, provided it can be demonstrated that as a surgical procedure thoracoscopy is free of serious accidents and complications.

In order to determine how safe thoracoscopy was, the records of all patients subjected to thoracoscopy without pneumonolysis at Olive View Sanatorium were reviewed, paying particular attention to the type of complication and its time relation to the operation. The period covered extended from 1930 to September, 1942.

Thoracoscopy alone, without pneumonolysis, should not be credited with complications arising as a result of postoperative "pushing" of the collapse by excessively large refills in the attempt to convert adhesions not feasible for pneumonolysis into feasible ones; this is particularly essential because this series of patients consisted of mechanically unsatisfactory pneumothoraxes, a group especially susceptible to complications.

Regardless of the conclusions derived from this study relative to the safety of thoracoscopy it is admitted that, if patients with adhesions of roentgenologically doubtful feasibility are subjected to thoracoscopy, the procedure does become dangerous in the hands of the easily tempted operator for no other reason than that a larger number of difficult pneumonolyses is bound to be done and followed by a greater incidence of serious complications. On the other hand, thoracoscopy in the case

From the Olive View Sanatorium, Olive View, California.
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of adhesions not feasible for pneumonolysis brings forth in an impressive way the ineffectiveness of the collapse and the danger and futility of maintaining it and thus often helps to bring about earlier abandonment of the ineffective collapse and substitution of an effective procedure.

PREOPERATIVE STATUS

This series consists of 73 tuberculous patients with mechanically unsatisfactory pneumothorax most of whom had sputum positive for tubercle bacilli and x-ray evidence of cavitation in the homolateral lung.

The type of infiltration in the homolateral lung was as follows: exudative in 42 patients, fibrotic in 1, and mixed exudative and fibrotic in 30. All but one were afebrile at the time of thoracoscopy.

The interval between the date of induction of pneumothorax and the date of thoracoscopy ranged from 1 to 32 months, and averaged 9 months. In 22 patients the interval was less than 6 months and in 49 less than 10 months. In 17 patients the interval was from 10 to 20 months and in 7 patients, 20 months and over.

Homolateral obliterative pleuritis with positive intrapleural pressures was present in 14 patients, 6 of whom also had a pleural effusion.

Homolateral pleural effusion existed in 15 patients, 6 of whom also had obliterative pleuritis, and was serous in 14 and purulent in 1. Of the 14 serous effusions, 9 were afebrile in onset and the remaining 5 had a febrile onset. Bacteriologic studies revealed that in the 9 cases in which the fluid was examined it was found to be negative for pyogens on culture. In 5 of the 9 cases examined the fluid was negative and in the remaining 4 positive for tubercle bacilli on culture; in only 1 was it also positive on smear. Of the 4 positive for tubercle bacilli the onset of effusion was febrile in 3; of the 5 negative for tubercle bacilli the onset was febrile only in 1.

OPERATION

The 73 thoracoscopies were done by five successive operators; I did only the last four during the year ending in September, 1942. Of this series, 5 patients were subjected to closed pneumonolysis some time prior to thoracoscopy and 3 other patients some time following thoracoscopy.

The duration of the operation ranged from 10 to 180 minutes and averaged 32 minutes for the 65 procedures for which this was recorded. The operation lasted 30 minutes or less in 43 cases, 60 minutes or less in the case of 59 patients, and over 60 minutes in 6 patients. These 6 prolonged procedures were done in the early 1930's and although the reason for it is not entirely clear in every case, technical difficulties with the thoracoscope accounted for prolongation of the operation. Pneumonolysis was not feasible in the case of all but 2 patients, and in these the procedure was limited to thoracoscopy because of severe dyspnea in one case and a cardiorespiratory reaction to locally injected novocain in the other.

POSTOPERATIVE COMPLICATIONS AND DEVELOPMENTS

The postoperative stay of these patients at the Sanatorium ranged from 2 weeks to 37 months and averaged 15 months. Eighteen patients stayed less than 6 months, 29 patients less than 10 months, 20 patients between 10 and 20 months, and 24 patients stayed 20 months or more.

Mortality attributable to thoracoscopy was zero. In the patients who died at the Sanatorium, death was due in 3 cases, 12 to 18 months postoperatively, to progressing pulmonary and/or intestinal tuberculosis, and in 1 case to a cerebral accident 4 months postoperatively. In 3 of these 4 patients the postoperative course was entirely uneventful and the remaining patient developed a persistent serous effusion 10 months postoperatively and died 4 months later of progressing pulmonary tuberculosis.

Morbidity following thoracoscopy will be discussed under the headings of hemorrhage, fever, spontaneous pneumothorax and bronchopleural fistula, pleural effusion, obliterative pleuritis, and extension of the pulmonary process, which is the approximate chronologic order in which accidents and complications of intrapleural operations occur.

Only 3 instances of hemorrhage could be found and all were slight; 1 was from the trocar wound externally and was described as due to failure to find the pleural space, and the remaining 2 were discovered in the course of postoperative aspiration of pleural fluid.

A temperature of 100° F. or over was arbitrarily chosen in this study to designate fever. Accordingly, 22 patients, or 30 per cent, were febrile. A maximum temperature of 100° F. was observed in 13 patients, 101° in 6, 102° in 1, and 104° in 2 patients. The duration of continuous fever ranged from 1 day to 20 weeks, an average of 2¼ weeks. The interval between thoracoscopy and the onset of fever was 0 in 19 cases, and 3, 6, and 44 weeks respectively, in the remaining 3 cases. All 3 patients developed a pleural effusion simultaneously with the onset of fever and all 3 received refills now regarded as too large; it is highly probable that thoracoscopy cannot be regarded responsible for these "late" complications.

Homolateral, serous, pleural effusion developed in 9 patients and persisted in 3 others who had it preoperatively as well. In 3 of the former it was transient and occurred within the first postoperative month, whereas in the remaining 6 it occurred in from 1½ to 10 months and persisted, eventually becoming purulent in 4 of these patients. On culture, tubercle bacilli were demonstrated in 1 of the 2 transient effusions and in all of the 6 persistent ones, 4 of the latter also containing tubercle bacilli demonstrable on smear as well. It will be noted that in 7 of the 15 patients with a preoperative effusion, thoracoscopy was followed by disappearance of the fluid.

All postoperative effusions with the exception of 2 purulent ones were negative for pyogens on culture, and the latter 2 were examined by smear only and were negative.

Spontaneous pneumothorax occurred in 3 cases in from 6 to 16 weeks postoperatively and was probably due to rupture of adhesions by refills of 500 to 600 c.c., currently regarded as heroic, in the effort to stretch the adhesions. A bronchopleural fistula developed in 1 case 3 days postoperatively; in this case the operation lasted over 2 hours and although it would seem that it was confined to inspection of adhesions, the record is not entirely clear on this point; moreover, 2 months earlier this patient was subjected to a closed pneumonolysis.

Obliterative pleuritis occurred in 8 of the 59 patients free of this complication preoperatively. Two of these left within 2 months and 1 had an open pneumonolysis 2 weeks following thoracoscopy; in 6 others pneumothorax was immediately discontinued. This left 50 patients with maintained collapse, 8 of whom developed obliterative pleuritis 6 to 40 weeks postoperatively.

The subject of extension of pulmonary disease cannot be discussed in the case of 15 patients either because they left the Sanatorium within 2 months postoperatively and were not x-rayed, or because pneumothorax was discontinued immediately. Of the remaining 58 patients, 5 showed definite contralateral extension of disease in from 1 to 10 months postoperatively; in 2 of these it was preceded by spontaneous pneumothorax or bronchopleural fistula and empyema.

RELATION OF OPERATION TO POSTOPERATIVE COMPLICATIONS AND DEVELOPMENTS

Fever.—The incidence of postoperative fever was found to bear practically no relationship to the length of the interval between thoracoscopy and induction of pneumothorax or to the presence of preoperative obliterative pleuritis. However, the type of infiltration in the homolateral lung and the duration of operation unquestionably influenced the incidence of postoperative fever; the latter was twice as great in those with exudative lesions as in those with mixed infiltration and two and one-half times as great in operations lasting over 60 minutes as in those lasting 30 minutes or less. It was rather surprising to find that postoperative fever was two and one-half times as frequent among those free of preoperative homolateral effusion as in those with one.

In 87 per cent of patients with postoperative fever its onset was immediately following thoracoscopy; it must be conceded, therefore, that postoperative fever was definitely due to the operative procedure. However, it is obviously not a serious sequel for the simple reason that the incidence of immediate fever of 102° F. and over, in the entire series, was only about 3 per cent.

Pleural Effusion.—The incidence of postoperative homolateral serous effusion showed the same relation to the type of homolateral infiltration and to the duration of pneumothorax as was observed in the case of fever. Among the patients with preoperative obliterative pleuritis the incidence of postoperative pleural effusion was three times as great as

among those without it. No relation was apparent between the incidence of postoperative effusion and the length of the operation.

The 6 postoperative effusions which appeared in from 1½ to 10 months postoperatively and which persisted cannot justifiably be regarded as due to thoracoscopy because of the length of interval and because of accompanying complications pointing to the presence of other factors such as "pushing" mechanically ineffective pneumothoraxes and subsequent rupture of adhesions. The 3 postoperative effusions which appeared within the first month and which were transient and unaccompanied by any complications were probably due to thoracoscopy. Among the 52 patients observed for two months and over postoperatively, free of serous fluid preoperatively and in whom pneumothorax was not discontinued, the incidence of postoperative effusion was only 17 per cent as compared to 20 per cent incidence preoperatively, which suggests that thoracoscopy did not contribute appreciably, if at all, to the incidence of this complication of pneumothorax.

Spontaneous pneumothorax, bronchopleural fistula, purulent pleural effusions and extension of pulmonary disease can be best considered together because of their associated pathogenesis in 5 out of the 7 patients falling into this group of postoperative developments. The fact that in most of these patients these complications occurred many weeks to a year postoperatively, the excessively large refills of air in those in whom spontaneous pneumothorax occurred and finally the 10 per cent incidence of these developments probably not exceeding the frequency with which they are encountered in similarly managed cases not subjected to thoracoscopy suggest that in this series thoracoscopy was not responsible for these complications. The reason for the recently reported incidence of bronchopleural fistula and empyema within 1 month following thoracoscopy alone, at the Sea View Hospital, is not understandable.[9, 11]

That *mixed infection* tuberculous empyema did not occur in this series lends weight to John C. Jones' statement that the usual source of pyogenic infection of the pleural space following intrapleural operations is traumatized lung rather than a "break in technique."[6]

Obliterative pleuritis postoperatively did not appear to bear any relation to the type of infiltration, duration of operation, etc. Among patients free of this condition preoperatively and observed for 2 months or over postoperatively the incidence of obliterative pleuritis was 16 per cent with an onset of 6 to 40 weeks postoperatively. It is stated that the incidence of obliterative pleuritis following closed pneumonolysis is definitely higher than in pneumothorax patients not subjected to the operation.[6] In this series where no division of adhesions was attempted the incidence of postoperative obliterative pleuritis was only 16 per cent as compared to an incidence of 20 per cent preoperatively, which suggests that thoracoscopy did not contribute appreciably, if at all, to the incidence of this complication.

Obliterative pleuritis with eventual loss of the pleural space following thoracoscopy of a patient with inoperable adhesions and ineffective pneumothorax may be regarded as a physiologic, and incidentally desirable, development or end-result of ineffective collapse by pneumothorax. In many instances it is undoubtedly a fortunate development terminating an ineffective procedure subject to serious complications such as spontaneous pneumothorax, bronchopleural fistula, or pyopneumothorax with loss of function of the entire lung including uninvolved parts, and homolateral and contralateral extension of disease.

SUMMARY AND CONCLUSIONS

The preoperative status, complications and developments following 73 thoracoscopies done by 5 successive operators at Olive View Sanatorium over a period of about 13 years are presented. Of all postoperative complications and developments observed, it appears that fever was the only one directly attributable to thoracoscopy and that pleural effusion and obliterative pleuritis could possibly be classified as true complications if it were not for the fact that their postoperative incidence, over a longer period of observation, was even lower than the preoperative incidence. Even if regarded as true complications, their incidence was not high so that it must be conceded that thoracoscopy, though a surgical procedure, was free of serious accidents and complications in this series. The limitations of roentgenography in studying pleural effusions, despite its definite usefulness in most cases, has already been pointed out.

Once it has been demonstrated that thoracoscopy is safe and not replaceable by roentgenography, the obvious conclusion is that thoracoscopy deserves a wider application in order to render ineffective collapse effective where pneumonolysis is feasible and to discontinue pneumothorax at an earlier date and substitute an effective procedure, where pneumonolysis is not feasible, before disastrous complications of ineffective pneumothorax supervene.

The clerical assistance of the Record Office staff of the Olive View Sanatorium is hereby gratefully acknowledged.

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PRIMARY FIBROSARCOMA OF THE CHEST WALL FOLLOWING THORACIC TRAUMA

JAMES M. BLAKE, M.D., AND JOSEPH K. BRADFORD, M.D.
SCHENECTADY, N. Y.

THE role of trauma in the causation of malignant tumors has been extensively discussed and is well recognized. This is particularly true regarding sarcoma of various types.

The proportion of such tumors originating in the chest wall is relatively small when compared to those occurring elsewhere in the body. Speed[1] states that the majority of malignant tumors of the chest wall following trauma occur in males, and that the most common site is the anterolateral chest wall, as this is the area most exposed to blows and to falls.

Hedblom,[2] in reporting sixty-one cases of sarcoma of the chest wall seen by him, or original records examined by him, states that thirteen occurred after trauma to the same area. Cohn,[3] has reported a case of fibrosarcoma of the chest wall, similar in many respects to the one presented here, in which surgical removal was possible.

The difficulty in establishing a definite causal relationship between trauma and tumor is obvious. In this connection five conditions postulated by Ewing would seem to meet any criticism. They are: (1) The authenticity and sufficient importance of the trauma must be established; (2) the previous integrity of the wounded part must also be established; (3) a reasonable time relationship of from three weeks to three years must elapse; (4) the identity of the injured area with the site of the tumor must be shown; (5) the nature of the tumor, as established microscopically, must be of a type that may be reasonably caused by trauma. The second of these conditions is usually difficult to fulfill inasmuch as the majority of patients have never had roentgenograms taken prior to the injury. In the case to be discussed this was true; but in a healthy boy 16 years of age a normal chest wall is the rule rather than the exception.

CASE REPORT

History.—P. N., a white male, aged 20 years, was referred to the consultation clinic of the Schenectady County Tuberculosis Hospital, by his family physician on April 12, 1940, and subsequently was admitted to the hospital on April 16, 1940. He complained of constant, dull pain in the right anterior chest at the anterior axillary line over the fifth and

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sixth ribs. He had been struck by an automobile in March, 1936, suffering fractures of the fifth and sixth ribs on the right. The clinical diagnosis was evident although no x-rays were taken. The ribs were strapped and apparently healed without incident, as he had no further symptoms. While playing sand-lot football in November, 1939, he was struck again a hard body blow at the site of his old injury. He continued his normal activities and did not seek medical aid at the time. Pain at the site of the injury persisted, however, and grew progressively worse in spite of strapping, causing loss of sleep and distress while working. There were no associated pulmonary symptoms. He had gradually lost forty pounds since November, 1939.

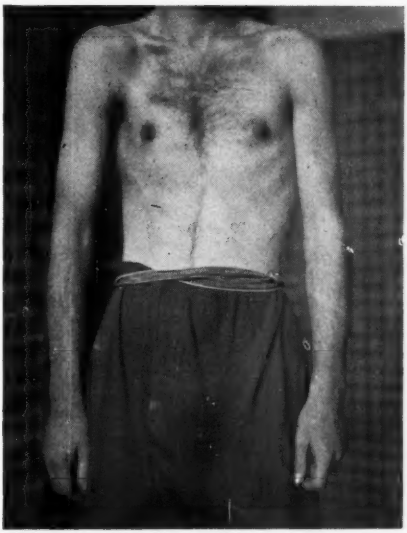

Fig. 1.

Physical Examination.—A well-developed young man was seen, apparently suffering pain in the right anterior chest, which was not influenced by motion or respiration. There were a few small palpable lymph nodes in the posterior triangles of the neck. The base of the neck appeared enlarged, particularly on the right, though no masses could be palpated. The external jugular vein on that side was unduly prominent. Moderate mid-dorsal scoliosis to the left was present. The right chest showed definite limitation of motion, and appeared flattened anteroposteriorly. The right shoulder was held high, and more than 90 degrees abduction of the right arm caused an increase in pain over the anterior chest. The infra- and supraclavicular fossae were increased with atrophy of the muscles about the shoulder girdle. In the anterior axillary line, over ribs five and six, there was a bulging mass measuring 5 by 7 cm., which was tender to slight pressure (see Fig. 1).

The sixth rib appeared to be irregular to palpation in this area. Hyperesthesia was present over the mass, and extended forward to the sternum. A firm cordlike structure could be felt running downward and anteriorly from the top of the axilla to the site of the mass, just beneath the skin. There was a well-marked area of diaphoresis involving the entire right posterior chest, beginning at the spine and

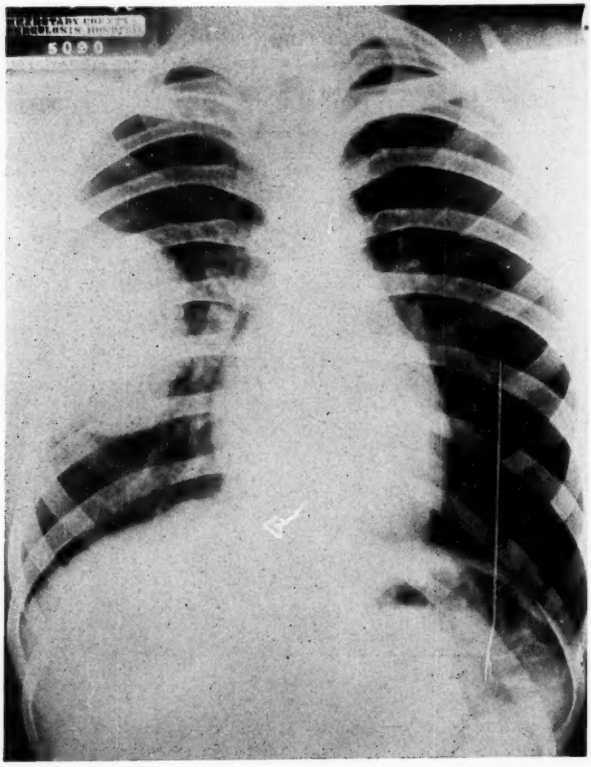

Fig. 2.

extending over the axilla and the lateral half of the right anterior chest wall. The axillary lymph nodes were not palpable. The percussion note was impaired, and breath sounds diminished, to the level of the eighth rib in the axilla, and also anteriorly below the third rib. Blood pressure in the right arm was 110/90 and in the left arm 90/60. There were no other abnormal physical signs.

Laboratory and X-ray.—The complete blood count, urine, sputum, and blood chemistry were within normal limits. The admission x-ray film showed a dense, oval, well-circumscribed mass, located laterally and anteriorly in the right chest, apparently adherent to the chest wall. It

measured 7 by 10 cm. and was between the third and seventh anterior ribs (Fig. 2). Destruction of bone in the sixth rib at the anterior axillary line was noted.

Clinical Course.—As a further aid in localization, a diagnostic pneumothorax was instituted on April 30, 1940, and subsequent chest x-ray films showed the tumor to be adherent to the chest wall, and apparently invaginating into the right lung (Fig. 3). X-rays of the skull, pelvis, and long bones showed no evidence of metastases.

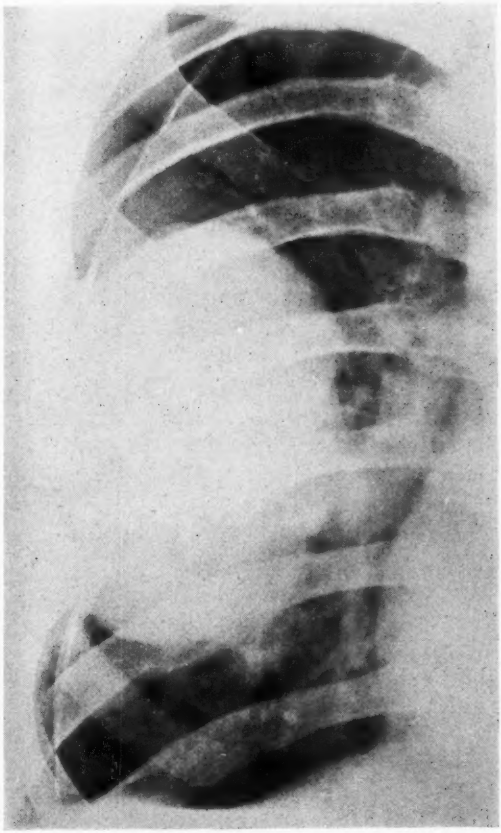

Fig. 3.

The pain was almost continuous and at times little relief was obtained except by fairly large doses of morphine. Weight loss continued at the rate of about two pounds each week.

Surgery seemed to be indicated and in view of physical findings a biopsy of the right axillary glands and the thrombosed vein in the right axilla was performed first. Although the glands showed marked

hyperplasia, no tumor infiltration was found. However, the lumen of the thrombosed vein was filled with elongated, slightly basophilic cells, showing numerous mitotic figures.

At the time of operation, May 11, 1940, it was found that the tumor had eroded the sixth rib and that there was extensive infiltration throughout the overlying musculature, extending posteriorly as far as the vertebral gutter. No removal was attempted, and after a biopsy of tumor tissue itself had been taken, the third, fourth, fifth, and sixth intercostal nerves were cut. The wound healed slowly, but firmly, and the pain was definitely less following the operation.

From May 27, 1940, to June 10, 1940, a course of deep x-ray therapy was applied to the right chest, a total of 3,900 r. units being given. This produced no appreciable effect on the size of the tumor or in relief of symptoms. Another course of deep therapy was begun in August, 1940, but was discontinued after two treatments because of increased pain, fever, vomiting, and generalized aching, lasting for two weeks.

He suffered with several attacks of bronchial obstruction with secondary suppuration, each lasting several days.

The patient expired on Aug. 8, 1941, twenty-one months after injury.

Essential Post-mortem Findings.—The sixth rib showed a transverse fracture in good position in the anterior axillary line. The ends of the fracture were covered by soft, grayish-white, ragged neoplastic tissue. The intercostal muscles in the vicinity of the fractured ribs were replaced by grayish-white neoplastic tissue extending to the vertebral column.

The middle and lower lobes of the right lung were markedly compressed, firm and noncrepitant. The cut surfaces were smooth and grayish white, and showed several well-circumscribed, firm white nodules varying in diameter from 0.7 to 2.5 cm. The lung tissue adjacent to the diaphragmatic surface was friable and contained many small abscess cavities filled with thick grayish-white pus. A soft oval nodule 1.8 by 0.9 by 0.9 occluded the lumen of the main stem bronchus of the lower lobe at a distance of 1 cm. from its junction with the main stem bronchus of the upper lobe. The nodule had a smooth surface and was attached to the bronchial wall by a broad pedicle. The distal portion of the occluded bronchus was filled with thick, yellow pus. The pleural cavity opposite the middle and lower lobes contained a firm, oval mass 12.5 by 8.5 by 3.5 cm. (Fig. 4). It was encapsulated except for an area 5 cm. in diameter adjacent to the fractured sixth rib where the neoplastic tissue was continuous with that found on the thoracic wall. The tumor tissue contiguous with the lung parenchyma was covered by tissue resembling thickened pleura. The parietal pleura covering the tumor mass was thickened, grayish brown, and opaque. Cut surfaces of the mass were smooth, brownish white, and coarsely lobulated. Stroma was not seen. One of the lobulations near the surface of the

tumor showed a large area of bright yellow and dark red discoloration. The mediastinal lymph nodes were enlarged, firm, and appeared to contain neoplastic tissue.

Microscopic Findings.—Sections from the large tumor of the right chest wall showed a compact mass of thick interlacing bundles of elongated fusiform cells, somewhat fibrillar in appearance. The cytoplasm in most of the cells was blue-staining. In some areas the cytoplasm was

Fig. 4.

pink-staining and hyalinization was present. In one section large areas of hyalinized connective tissue were present containing abundant calcareous granules. Scattered about were small numbers of large mononuclears containing coarsely granular, golden yellow pigment. Some areas contained polygonal cells, loosely grouped together. Throughout the tumor, mitotic figures were present and vascularization was poor.

Sections from the fractured ends of the sixth rib showed compact tumor tissue consisting of fusiform cells with an occasional small area of hemorrhage. Numerous small foci of osseous tissue were present.

DISCUSSION

This case shows clearly the necessity for more careful examination, including x-ray films, of patients who complain of: (1) Persistent pain at the site of an injury over a period of time considerably longer than that necessary for healing of the injury to take place; (2) the appearance of a mass at the site of an old injury; (3) the recurrence of pain at the site of an old injury, after a lapse of months, or years, when no recent cause is apparent.

The fact that these tumors of low-grade malignancy do occur in young people following what are often thought of as minor injuries should be kept in mind always. Most often the patient must shoulder the responsibility if an early diagnosis is not made, because he does not seek medical advice for such indefinite symptoms. However, no such complaints as those mentioned above should be dismissed with directions for symptomatic treatment when the physical examination alone shows no evidence of definite lesion.

Tumors of the chest wall such as fibrosarcoma are operable if diagnosed early. They metastasize slowly and most often by direct extension; and even after a considerable period of time has elapsed, excision of the tumor along with extensive dissection of the chest wall has proved successful. As was contemplated in this case, this operation can be combined with partial or total removal of the adjacent lung if it is involved.

The use of pneumothorax as a diagnostic aid in cases of chest wall tumor is very helpful. As shown in the case presented above it will aid in localizing the tumor mass more exactly and will demonstrate the site of origin of the tumor. Pneumothorax is also valuable as a preoperative procedure in these cases, especially when removal of the lung is contemplated.

X-ray therapy has been advocated for the treatment of malignant tumors of the chest wall, but most reports regarding this method of treatment are not encouraging. Deep x-ray therapy did not seem to alter the course of the process in this case and it would seem that malignant tumors of the thorax should be considered as surgical diseases.

One other incident seems worthy of comment. The thoracotomy incision was made through subcutaneous and muscle tissue widely infiltrated with tumor tissue. It has always been our impression that such incisions healed poorly, if at all, but in this case healing was prompt, by primary union, and remained firm.

SUMMARY

A case of primary fibrosarcoma of the chest wall following thoracic trauma is presented.

The difficulty of establishing trauma as an etiologic factor is discussed briefly and five conditions are given which, if fulfilled, should definitely establish the relationship.

The necessity for a complete examination, including x-ray films of the affected part, is shown in cases with persistent or recurring symptoms at the site of an old injury.

The value of pneumothorax as an adjunct in proper localization of a tumor mass in the chest wall is shown.

Tumors of the thorax would seem to be surgical entities and should first be considered as such.

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CONGENITAL CYSTS OF THE MEDIASTINUM

Report of Three Cases Including a Gastric Cyst

Herbert A. Carlson, M.D.
Minot, N. D.

AN EXTRAORDINARY variety of cysts, neoplasms, and inflammatory diseases occurs in the mediastinum. The cysts may be congenital, inflammatory, parasitic, or of undetermined origin. The discussion that follows will be concerned only with the congenital cysts, a group of cysts and cystic tumors believed to be the result of some aberration in embryologic development. A relatively rare group of cysts lined with epithelium derived from entoderm will be stressed. These "entodermal cysts" have sometimes been regarded as teratomas with a one-sided development but, as will be pointed out, they differ from teratomas, not only histologically, but as to location, pathogenesis, and clinical course.

Congenital cysts known to occur in the mediastinum are listed below:

1. Epidermoid cysts—derived from ectoderm
2. Dermoid cysts—derived from ectoderm and mesoderm
3. Teratomas—derived from all three primitive germinal layers
4. Cystic lymphangiomas—derived from mesoderm
5. Pericardial coelomic cysts—derived from mesoderm
6. Bronchial (bronchogenic) cysts—derived from entoderm and mesoderm
7. Gastric (gastrogenic) cysts—derived from entoderm and mesoderm
8. Enteric cysts (enterocystom)—derived from entoderm and mesoderm

It is quite probable that the above list does not include every type of congenital cyst that may be found in the mediastinum. Furthermore, it is recognized that some of the cysts are atypical and are difficult to classify; for example, those reported by Stoeckel and by Guillery may be examples of esophageal cysts, or they may be variations of the group referred to as bronchial.

EPIDERMOID CYSTS, DERMOID CYSTS, AND TERATOMAS

Although Hedblom made a distinction between epidermoid cysts, dermoid cysts, and teratomas, they are not separated by a sharp line of demarcation, and as they all have one feature in common, namely, that that they contain tissue derived from ectoderm, there has been a tendency to include the entire group under the one term, dermoid or

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teratoma. The pathologic and clinical features of teratomas have been described in some detail by Hedblom, Harrington, Heuer, and Andrus. These cysts or tumors occur as a rule in the anterior mediastinum, from which attachment they may, however, protrude laterally and posteriorly to occupy a major portion of either thoracic cavity or they may extend from the superior mediastinum into the base of the neck. They may grow to attain huge dimensions. A typical cyst is lined by stratified, squamous epithelium and contains hair and sebaceous material, but there are great variations in the gross and microscopic appearance and the mass may contain calcifications, cartilage, bone, teeth, muscle, glands, and other complex structures. Some teratomas are malignant.

Two theories have been proposed to explain the genesis of teratomas, the bigerminal and the monogerminal theories. According to the bigerminal theory, a secondary anlage develops in the body either through the fertilization of a polar body or the independent development of a blastomere. The monogerminal hypothesis assumes that there is an abnormal separation or segmentation and inclusion of cells, the morphologic differentiation of which depends on the stage in the embryo in which the separation occurs. The origin of most mediastinal teratomas is believed to be in the region of the branchial clefts where the embryonal anlagen are subject to considerable involution, the abnormal cellular inclusions being carried into the anterior mediastinum with the descent of the heart.

One of the important clinical features of teratomas is the absence of symptoms over a period of many years while the cyst is undergoing progressive growth and expansion. Heuer and Andrus found in reviewing 217 cases that only 5.5 per cent were discovered under the age of 12 years. As a rule, symptoms develop first at the age of 20 to 40 years. Pain, cough, and dyspnea are noted most frequently. Occasionally there is expectoration of hair, blood, or other material from the cyst. X-ray examination usually reveals a rounded nonpulsating mass in the anterior or superior mediastinum or extending into the lung field.

CYSTIC LYMPHANGIOMAS

The cystic lymphangiomas are among the less common of the mediastinal cysts and are characterized by the formation of multiple cysts. The case reported by Skinner and Hobbs occurred in a child of 7 years and the cyst was located extrapleurally on the right side attached to the pericardium, inferior vena cava, diaphragm, and thymus. The cysts varied from the size of a pinhead to 2 inches in diameter, contained a clear, pale, or dark brown fluid, or gelatinous material, and were lined by a single layer of endothelium. The septae contained fat cells, foci of lymphocytes, blood vessels, smooth muscle, and cholesterol crystals.

In discussing the pathogenesis of cystic lymphangiomas, Skinner and Hobbs suggest that they may be derived from a portion of the anlage for

vessel formation drawn down from the region of the gill clefts by the pericardium in its descent. A thymic origin has also been considered a possibility.

The symptoms produced are not pathognomonic of this type of cyst, but the onset of symptoms is probably earlier than in teratomas.

PERICARDIAL COELOMIC CYSTS

The pericardial coelomic cysts are simple thin-walled cysts. Two cases were reported by Lambert, each found at operation lying directly in contact with the anterior chest wall, diaphragm, pericardium, and lingula of the left lung. The cysts were lined by endothelium or mesothelium resting on a loose-textured fibrous tissue with a number of capillaries, some fat, and a few extravasated blood cells. Such a cyst, Lambert believes, develops from one of the several primitive pericardial lacunae. One of the lacunae fails to merge with the others, persists, and develops into an independent cavity, forming a cyst.

Both of Lambert's cases were asymptomatic and were discovered during routine examinations.

BRONCHIAL CYSTS

Ciliated columnar respiratory epithelial cysts, known also as bronchial or bronchogenic cysts, may contain all of the tissues found normally in the bronchial wall. They are lined typically by ciliated columnar epithelium although in some areas or in some cysts the cilia may be absent. Some cysts that probably belong to this group are lined by cuboidal epithelium. Complete loss of epithelium over large areas may occur. The wall may contain mucous glands, fibrous tissue, elastic fibers, smooth muscle arranged in layers, cartilage, nerves, and blood vessels. Cartilage or other elements of the bronchial wall may be absent. They are cysts of the posterior mediastinum or the posterior portion of the superior mediastinum and are found usually in the neighborhood of the bifurcation of the trachea. They may extend upward for some distance between the esophagus and trachea or, occasionally, anterior to the trachea, and they often compress the trachea or one or both main bronchi. As a rule, the lumen of the cyst does not communicate with the bronchi or the trachea. Most of the cysts described have measured 5 cm. or less in diameter and have contained 50 c.c. or less of clear, viscid, mucoid fluid, sometimes described as having the appearance of white of egg. Cysts of this type have been reported by Blackader and Evans, Mixter and Clifford, Johnston, Rizzi, and Alford. Additional cases are included in the case records of the Massachusetts General Hospital and in reviews of the literature by Johnston and Heuer and Andrus.

The genesis of bronchial cysts has been explained on the basis of a pinching off of a diverticulum of entoderm and mesoderm from the foregut in the region of the tracheal bud, or a secondary budding of the

tracheal bud itself, or an abnormal division in a later stage of development of the tracheobronchial tree. Another theory assumes an imperfect closure during fetal life of the communication between the trachea and esophagus.

Bronchial cysts by obstructing the air passages may produce symptoms early in life before the cyst has attained great size. In this respect, they contrast sharply with the teratomas. Occasionally, the cyst may remain asymptomatic throughout life as in the autopsy case reported by Rizzi in a man of 66 years, but more commonly the symptoms first appear in the newborn period or in the first few months of life. The infant may be stillborn apparently because of the cyst. Dyspnea, which is the most common symptom, may be continuous or it may occur in attacks sometimes associated with cyanosis. Frequently, the infant presents evidence of dysphagia by taking his feedings poorly or by crying after attempting to eat. Cough, which may be croupy or brassy, is a common symptom and atelectasis and pneumonia often supervene.

The frequent occurrence of atelectasis makes the diagnosis difficult as the physical signs are those of the complication and the relatively small cyst cannot readily be visualized on the x-ray film, especially when surrounded by atelectatic or consolidated lung tissue. Presumably bronchography and bronchoscopy have been employed only rarely in the diagnosis of bronchial cysts although these methods, particularly bronchoscopy, should contribute to the diagnosis by demonstrating narrowing of the bronchi due to external pressure.

GASTRIC CYSTS

Gastric (or gastrogenic) cysts reproduce the structure of the stomach wall. A gastric mucosa with deep branching glands, parietal and chief cells was described by Mixter and Clifford. The wall may contain muscularis mucosa, submucosa, musculature consisting of circular and longitudinal layers, and a subserosa with arteries and veins. The cyst is partly covered by pleura. Böss demonstrated nerves and ganglion cells. There may be a loss of mucosa over extensive areas and Böss and Seydl have each reported a chronic peptic ulcer occurring in a gastrogenic cyst with perforation into the lung and fatal pulmonary hemorrhage.

The cysts may vary greatly in size. They may contain only a few cubic centimeters or as much as 400 c.c. of fluid, which may be milky or clear amber or sanguineous in appearance, viscid, and either acid or neutral in reaction.

The cyst is situated posteriorly in the mediastinum either on the right or left side usually behind the root of the lung and adherent to the lung and mediastinal structures. Attachment to the diaphragm was noted in the case reported here, and one of the cysts reported by Mixter and Clifford appeared to be partially imbedded in the diaphragm. Mixter and Clifford also reported a multilocular cyst behind the trachea and esophagus causing erosion of the vertebrae and ribs.

The genesis of gastrogenic cysts has been ascribed to the pinching off of a bud or diverticulum of the embryonic foregut, to an entodermal epithelial germ cell of the esophagus capable of producing gastric mucosa, or to an intrathoracic vestige of the ductus omphalomesentericus.

The symptoms of gastric cysts are much the same as those of the bronchial cysts. Dyspnea, cyanosis, cough, and dysphagia are prominent, occurring as a rule in the first few weeks or months of life. Respiratory infections are frequent complications.

As the cyst may attain a substantial size rather early, physical signs may be more helpful than is the case with bronchial cysts. Dullness or flatness and decrease of the breath sounds, usually on one side posteriorly, suggest the presence of fluid. Although fluid may be present, these signs may be due to the cyst alone. X-ray examination may reveal a rounded mass in the mediastinum or in either lung field or the outline may be indistinct due to the presence of fluid or atelectatic lung tissue. Lateral films are of value in demonstrating the posterior position of the cyst.

Aspiration of the pleural fluid and the production of a small pneumothorax may enhance x-ray visualization. In the case reported here (Case 3), aspiration of the cyst itself provided the clue to diagnosis as the fluid was viscid, unlike ordinary pleural fluid. The aspiration of fluid containing hydrochloric acid is practically diagnostic of a gastric cyst, but the acid may apparently be neutralized by other secretions of the lining of the cyst.

ENTERIC CYSTS

Cysts resembling gastric cysts except that they are lined by intestinal mucosa have been reported in the German literature under the term, "enterocystom." Only three cases could be found in the literature by Brass to which he added one of his own. Black and Benjamin have reported a case of "enterogenous cyst" of the mediastinum in an infant who also had a perforated peptic ulcer of an "intramesenteric reduplication intestinal pouch." In their case, the lining of the mediastinal cyst was undeveloped or destroyed and it is impossible to prove that the cyst was a purely enteric cyst. It may have been either enteric or gastric.

One of the cysts reported here (Case 3) was at one time believed to be an enteric cyst. Ultimately, it was demonstrated that in some areas the epithelium contained acid secreting cells. To complicate the picture further, areas of stratified squamous epithelium were also present. Apparently, the gastric and enteric cysts are so much alike that their differentiation may depend on careful study of microscopic sections prepared from several portions of the wall of the cyst. Furthermore, areas of epithelium may be present which are more characteristic of the esophagus than of either stomach or bowel. Perhaps it is futile to attempt to make all cases fit into an arbitrary classification.

The pathogenesis of enteric cysts has been explained by two theories; that they arise from the foregut or that they represent rudimentary segments of the thoracic portion of the omphalomesenteric duct. Roth's case in which a retroperitoneal and an intramesenteric cyst were associated with an intrathoracic cyst has been cited in support of the latter theory. All three cysts occurring in one individual, reproducing the structure of the intestine, leads to the assumption that all three had a common origin from the omphalomesenteric duct. On the other hand, multiple anomalies may occur in one individual and the complex nature of some of the cysts (not purely intestinal cysts) is best explained by the theory that they are derivatives of the foregut.

The four (or five?) cases of enteric cysts reported to date have all occurred in infants. All were observed at autopsy. None were operated upon.

CASE REPORTS

The following case reports include a teratoma, a bronchial cyst, and a cyst which is somewhat atypical but is of the gastric type. The bronchial cyst was observed at autopsy, the others were removed at operation.

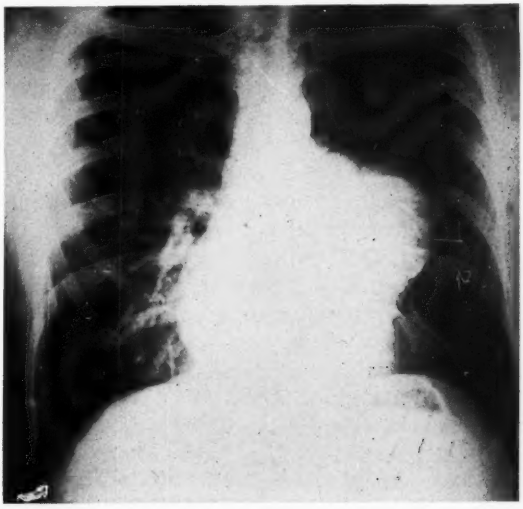

Fig. 1.—Chest film (Case 1). The mass extends from the mediastinum into the left lung field.

CASE 1.—S. H., the patient (referred by Dr. G. S. Seiffert), was a 23-year-old college student who had no complaint but was required to have a routine physical examination. He was well developed, well nourished, and no abnormal physical signs were noted. Hemoglobin was 96 per cent; leucocyte count, 7,150. Because of a positive Mantoux test, an x-ray of the chest was requested.

An x-ray film on April 22, 1942, showed a large tumor mass extending out from the left hilar region (Fig. 1). Lateral films showed this tumor to lie in the anterior portion of the mediastinum. The anterior margin of the tumor was directly behind the anterior chest wall. The tumor measured 10 cm. in diameter in the lateral view. On the basis of the x-ray findings, the diagnosis was mediastinal tumor, probably dermoid cyst. The patient was advised that because of the danger of complications and the possibility that the tumor might be malignant, it should be removed.

The operation was performed on May 16, 1942, under nitrous oxide, oxygen, and ether. An anterior approach was employed, the skin incision being made along the fourth interspace with a perpendicular arm extending upward to the second costal cartilage. The pectoralis major muscle was incised in the same region and separated off the ribs. The fourth costal cartilage and anterior portion of the rib were resected. An incision was made into the periosteal bed to enter the pleural cavity. About 1 cm. of the third and second costal cartilages was excised near the sternum and the pleural incision continued into this region. A cystic mass of the anterior mediastinum was found adherent to the lung and pericardium and embedded in the mediastinum. The main portion of the cyst was freed from these structures by sharp and gauze dissection. There remained a stalk in the superior pole of the cyst which extended upward and deeper into the mediastinum. The cyst, together with the nodular stalk, was excised. The base of the stalk was transfixed and ligated after making reasonably sure that no tumor tissue remained. The lung was inflated from time to time with positive pressure and again at the end of the operation. Closure was made with interrupted mattress sutures in the periosteal bed and continuous sutures in the anterior limb of the incision. The pectoralis major muscle was then sutured over this line of suture with continuous chromic catgut. The skin was closed with continuous dermal. The patient's condition remained satisfactory throughout.

Postoperatively, the patient was placed in an oxygen tent. He received two blood transfusions although his blood pressure remained normal throughout the postoperative period. Some serosanguineous fluid accumulated in the pleural cavity and was treated by repeated aspirations. The patient was discharged from the hospital on the twenty-eighth day. He still had a small pleural effusion which has since disappeared.

The pathologist's report (Dr. P. J. Breslich) is summarized as follows: This cystic structure is about 10 cm. in diameter. On surfaces made by cutting, the lumen of the cyst is found filled with desquamated epithelium and masses of blonde hair. In one place in the cyst wall is a papular structure about 3 cm. by 2½ cm. The structure is apparently covered by stratified squamous epithelium to which are attached blonde hairs. In the base of the cyst, beneath the pedicle of the

papular structure, is dense fibrous tissue with cystlike places that vary from 1 mm. to 5 or 6 mm. in diameter. This fibrous tissue mass is about 3½ mm. in diameter and 2 cm. thick (Fig. 2).

Histology.—The wall of the mediastinal cyst consists chiefly of densely hyalinized fibrous scar. Between the bundles of scar tissue are spaces containing huge vacuoles evidently foreign material, possibly from the lumen of the cyst. Immediately surrounding the vacuoles is chronic granulation tissue densely infiltrated with small round cells. In the

Fig. 2.—Teratoma (Case 1) preserved in formalin. The cyst is open at one point and hair from the lining has been extruded.

nodule in the wall of the cyst are glandular structures. The glandular structures are lined by a high columnar epithelium. The epithelial cells have basal nuclei often arranged in two or three layers as in pseudostratified columnar epithelium. The contents of these glandular structures are pink and granular. In some of the sections, the columnar epithelium is definitely ciliated. The columnar epithelium frequently is arranged in papillary folds. Near some of the glandular structures are

masses of lymphocytes, possibly of an inflammatory nature, but occasionally there are definite lymph follicles. The lining of the large cyst is a stratified squamous epithelium covered with masses of desquamated cells. Beneath the epithelium in the nodule in the lining are masses of sebaceous glands and hair follicles.

Diagnosis: Teratoma of anterior mediastinum.

CASE 2.—This 8-month-old male infant was a patient of Dr. M. W. Garrison, who kindly permitted me to include his case.

The infant was admitted to Trinity Hospital on Nov. 2, 1936. The grandmother stated that the infant had had a cough thought to be croup for nearly four months. However, in spite of the cough the patient had continued to gain weight normally and had no fever. For two weeks the cough had been becoming more severe. On the day before admission to the hospital, the child seemed to be acutely ill for the first time. He vomited repeatedly. On physical examination, he was found to be well developed and well nourished. He was having difficulty in breathing and the respirations were noisy. The percussion note was found to be normal on both sides. Numerous coarse rhonchi were heard over both lung fields anteriorly and posteriorly. Otherwise the physical examination was negative. Hemoglobin was 58 per cent, the red blood cell count was 4,130,000, the white blood cell count was 5,300, and the urine was normal. The temperature on admission to the hospital was 102.2° F., the pulse, 170, and the respirations, 48. The temperature was septic and varied between 101 and 106.2° F. Pulse and respirations remained rapid. The patient became progressively worse and died on Nov. 5, 1936, seventy-two hours after admission to the hospital.

The post-mortem examination was performed by Dr. P. J. Breslich. His report is summarized below:

The body was that of a male white child, 65 cm. long and weighing about 25 pounds. Aside from the fact that the stomach and bowel were considerably distended with gas, the chief findings were those of the thoracic viscera. The lining of the trachea and main bronchi were slightly red and roughened. In the mediastinum, there was a hard cystic mass 3 cm. by 2 cm. by 1½ cm. which markedly compressed the trachea and main bronchi at the bifurcation of the trachea. It was situated anterior to the trachea and the right main bronchus. The arch of the aorta passed anterior and to the left of the mass and the superior vena cava passed anterior and to the right. It was easily stripped out from the mediastinal tissue and was firmly adherent to the trachea only at one point, 1.5 cm. proximal to the bifurcation and a little to the right of the midline. In this place the cyst was attached by a dense fibrous scar resembling a pedicle about 0.3 cm. in diameter and containing a few tiny nodules of cartilage. The lower portion of the trachea for about 1½ cm. proximal to the bifurcation, particularly on the right side, was compressed by the mass which also caused a marked compression and back-

ward curving of the right main bronchus. The cartilaginous rings of the trachea and bronchus in this region when viewed from the outside were concave instead of convex. After removal of the heart, it was found that the left main bronchus was also markedly compressed at its origin. The heart weighed 50 Gm. and was normal. The lungs together weighed 150 Gm. The tissue in the anterior portion of the lungs was bright yellow and crepitant. The alveolar spaces were widely distended with air. In the posterior portion of the lungs the tissue was dark red, nodular, and airless. The mediastinal mass was fixed in formalin and when opened was found to be a thin-walled cyst filled with gelatinous material.

Microscopic examination of the sections obtained from the posterior portion of the lungs revealed that the alveoli for the most part were irregular and partially collapsed. They contained dense exudates of large round cells and polymorphonuclear leucocytes. These exudates also packed the lumina of the smaller bronchi.

The lining of the cyst was a pseudostratified high columnar epithelium arranged in folds such as are frequently found in the lining of a contracted bronchus. The epithelium was distinctly ciliated. The submucosa contained a thin band of smooth muscle. It also contained masses of mucous glands and occasional sections of cartilage. The epithelium, mucous glands, and cartilage were arranged in such a way as to suggest a dilated bronchus.

The pathologic diagnosis was bronchial cyst of the mediastinum with marked compression of the trachea and main bronchi; marked acute emphysema of the anterior portion of both lungs; acute bilateral hypostatic bronchopneumonia; acute catarrhal tracheobronchitis.

Case 3.—D. H., male, aged 4 months, was seen by Dr. R. E. Dyson on Nov. 12, 1940. The chief complaint was rapid breathing, which the mother had noticed since the first week of life. Breathing at times was labored, the infant took his feedings poorly, and the mother thought that he had difficulty in swallowing.

On physical examination he appeared somewhat pale, and the nutrition was only fair. The weight was 10 pounds, 14¾ ounces. The body length was 24 inches. There was some bulging of the left side of the chest. Respirations were rapid, with some evidence of labored breathing. There was hyperresonance of the left anterior chest, flatness of the left upper chest posteriorly, with somewhat more resonance of the left lower chest posteriorly. The right side was normal to percussion. The breath sounds were normal on the right side, harsh over the left chest anteriorly, and very distant over the left chest posteriorly. The point of maximum cardiac impulse was on the right side. The hemoglobin was 74 per cent, leucocytes, 15,900, and the urine was normal.

A chest film on Nov. 12, 1940, was taken with the following report: "Fluoroscopic examination and a single film of the chest show the bones

to appear normal. The heart and mediastinal contents are displaced toward the right. Most of the heart lies in the right side of the chest. There is bilateral pneumothorax, with partial collapse of the lungs. There is some fluid in the left pleural cavity. Conclusions: Bilateral pneumothorax, plus fluid on the left side of the chest."

A needle was introduced into the left chest posteriorly. The fluid removed was thin and pale yellow. Cultures showed no growth on blood agar or in broth in forty-eight hours. The specific gravity was 1.010. Microscopic examination of the sediment showed five to fifteen pus cells per high-power field and twenty to thirty red blood cells. Because the mother had had pulmonary tuberculosis, some of this fluid was injected into a guinea pig. The guinea pig examination was negative. The aspiration was repeated. It was then noted that the fluid was viscid. A test with litmus paper was neutral in reaction. A diagnosis of intrathoracic cyst was made.

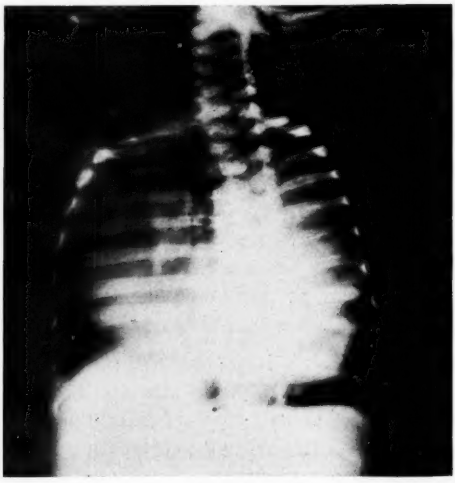

Fig. 3.—Chest film of infant (Case 3) taken after first aspiration, showing large cyst displacing the heart to the right. Only a small area of lung tissue is visible on either side.

Chest films taken on Dec. 2, 1940, were reported as follows: "Reexamination of the chest shows the heart to be displaced into the right thoracic cavity. There is some pneumothorax on both sides of the chest.* On the left side there is a large, well-circumscribed mass, which is denser than lung tissue and almost fills the left side of the chest. This probably represents a large cyst." (Fig. 3.)

At this time, because the baby was having attacks of dyspnea and also some difficulty in swallowing, these aspirations were repeated once every four to seven days. The amount of fluid removed varied from about 100 to 185 c.c. The dyspnea was not permanently relieved, how-

*Pneumothorax? Emphysema?

ever, and the difficulty in deglutition was increasing, so due to the fact that the child apparently was not in good enough condition for surgical removal of the cyst, an attempt was made on Jan. 24, 1941, to exteriorize the cyst. At this stage a rib resection was performed. The pleural cavity was opened. There were about 10 c.c. of dark, sanguineous fluid in the pleural cavity. A cyst was found extending throughout the lower two-thirds of the chest. It was smooth, very dark blue in color, with large blood vessels on the surface. It was attached to the chest wall near the spine but not at the site of the pleural incision. The cyst was sutured into the opening in the periosteal bed. It could not be brought out to the skin.

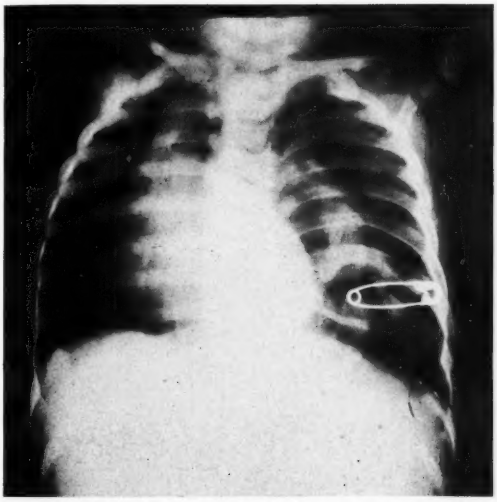

Fig. 4.—Chest film of same infant (Case 3) taken after drainage of the cyst. The cyst is strikingly reduced in size; the lungs have expanded.

One week later, the wound was reopened and the cyst which was now adherent was drained and a tube was inserted. The open edges of the cyst were sutured to the chest wall to strengthen the line of adherence. After drainage of the cyst, the patient's condition improved markedly. He gained in weight, became well nourished, and had no further attacks of dyspnea, but he was anemic, the hemoglobin varying from 41 to 69 per cent. Attempts to destroy the lining of the cyst with phenol and alcohol and with Carnoy's solution were unsuccessful (Fig. 4).

During the hot weather of the summer, it became difficult to protect the skin about the edges of the draining sinus. The skin became excoriated and although a variety of dressings were tried, it was impossible to keep the skin in normal condition. The appearance was very much like that of the skin about an enterostomy.

On Sept. 22, 1941, removal of the cyst was accomplished. The child now was 14 months old. Under nitrous oxide, oxygen, and ether an-

esthesia, a curved posterior incision was made, excising the fistula and resecting the regenerated rib. An intercostal incision was made in the seventh interspace and three ribs were divided posteriorly. Satisfactory exposure was obtained. The cyst was found adherent to the posterior chest wall, to the mediastinum, above and below, and behind the root of the lung, to the upper and lower lobes of the lung, to the diaphragm and pericardium. No stalk of any kind was observed. The lobes of the lungs were pushed anteriorly by the cyst. When the cyst had been removed, there remained a raw area in the mediastinum, and there were numerous divided adhesions, some delicate, some dense, which remained

Fig. 5.—Low-power photomicrograph of the wall of the cyst (Case 3) showing mucosa, submucosa, musclaris, and subserosa.

on the diaphragm, the pericardium, the lungs and the parietal pleura. The wound was closed and a tube inserted into the pleural space with water seal drainage. A blood transfusion was started before the incision was made. The postoperative course was satisfactory.

The patient was discharged from the hospital on Oct. 15, 1941. There was only a small area of drainage at the site of the tube which had previously been removed. This healed rapidly and the patient now is in excellent health. Postoperative films of the chest show that the heart is practically in normal position and the lungs have re-expanded.

The pathologic report by Dr. P. J. Breslich is as follows:

"This collapsed cyst is approximately 8 cm. long and 5 cm. in diameter. The outside is reddened and roughened with bands of recently cut fibrous tissue. The lining is finely nodular. The nodules vary from less than 1 mm. to as much as 3 mm. in diameter, and consist of dense fibrous tissue-like scar. The average thickness of the cyst wall is about 0.6 cm. The wall of the cyst contains fibrous nodules as much as 1.5 cm. in diameter. One of these, when cut into, contains a cyst about 0.3 cm. in diameter lined by a mucosa.

Fig. 6.—Higher magnification of the lining to illustrate the gastric mucosa (Case 3).

"*Histology.*—The lining of the cyst for the most part resembles mucosa from the small bowel. The wall is markedly edematous and diffusely infiltrated with small round cells. There are two distinct smooth muscle layers, one of them apparently circular and the other longitudinal. Beneath the mucosa are masses of glands resembling duodenal glands. Portions of the lining consist of stratified squamous epithelium lined by columnar epithelium. In a few places, the lining resembles stomach mucosa in which the glands are supplied with parietal cells with highly acidophilic cytoplasm.

"Diagnosis: Mediastinal cyst (embryonal rest of primitive gut)."

Some of the microscopic sections were also seen by Dr. E. T. Bell, head of the Department of Pathology, University of Minnesota. His first

impression was that the structure of the cyst was that of the intestine. However, when additional sections were prepared he reported as follows: "This has the complete structure of the body of the stomach including parietal cells. It is not like the antrum." (Figs. 5 and 6.)

SURGERY OF MEDIASTINAL CYSTS

The surgical management of teratomas has been well covered in the papers of Hedblom, Harrington, and Heuer and Andrus. It is agreed that exteriorization and drainage of the cyst should be avoided as a rule and that the ideal operation consists of complete transpleural removal of the tumor in one stage. There are some minor differences of opinion concerning details of the operation. Harrington favors a posterior incision in most cases. Heuer and Andrus state that an anterior

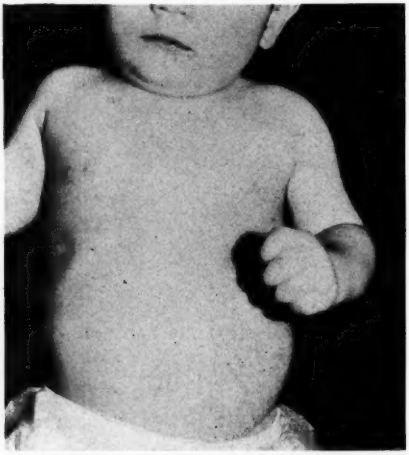

Fig. 7.—Anterior view of infant after operation (Case 3).

approach is applicable to tumors of small or medium size situated in the anterior mediastinum. In the removal of the teratoma reported here (Case 1) an anterior incision was found to be entirely satisfactory but probably not superior to a posterior approach. For a larger cyst a posterior incision would, undoubtedly, be advantageous.

Closure without drainage is favored by most authors. The fluid that accumulates postoperatively is removed by repeated aspirations. When infection is feared at the time of operation, closed intercostal drainage through a separate stab wound is recommended.

The teratomas including the epidermoid and dermoid cysts, being the group of cysts occurring most commonly in the mediastinum, are naturally the ones that have been operated on with the greatest number of successes. Another factor favoring surgery in the case of the teratomas is that the patient is frequently a relatively good surgical risk.

Hedblom in 1933 in reporting on 185 cases collected from the literature found that 99 patients had been subjected to a variety of operations and complete extirpation had been performed in 52. Improved results are to be found in more recent reports. Harrington, for example, operated on 16 patients with teratoid tumors. Two died in the postoperative period, one within six months, and thirteen were living and completely relieved of symptoms. Two of the three patients who died had malignant teratomas.

The surgical principles outlined previously apply in general to lymphangiomas and pericardial coelomic cysts, but in the surgical treatment of bronchial, gastric, and enteric cysts additional factors complicate the problem. Many of these cysts occur in infants who may die or develop pneumonia or other serious complications before the diagnosis can

Fig. 8.—Posterior view of infant after operation (Case 3).

be made. When the diagnosis is obvious, the infant may be a poor surgical risk because of malnutrition, cyanosis, dyspnea, and dysphagia, and the surgeon is confronted with the dilemma of performing a major thoracic operation on a sickly infant or exteriorizing and draining the cyst preliminary to excision. Some cysts particularly those of the bronchial type may be so situated within the chest as to make drainage impracticable. In that event, primary excision offers the only hope of cure. Experience with a gastric cyst (Case 3) indicates that drainage may sometimes be a very valuable, perhaps lifesaving, procedure. Mixter and Clifford, likewise, employed drainage in a similar case with satisfactory results.

It must be admitted that while drainage of a cyst relieves dyspnea and dysphagia, it may also create new problems. Excoriation of the skin has been mentioned (Case 3). It was feared also that preliminary drainage would multiply the dangers of infection of the pleura and mediastinum at the time of the final operation but a limited experience suggests that the potential danger of infection may be overcome by employing water seal drainage.

In brief, the one stage operation is ideal, but preliminary drainage has given sufficiently good results to justify further use of the method, especially for infants with dyspnea, cyanosis, or dysphagia.

The majority of the bronchial and gastric cysts and all of the enteric cysts reported thus far have been autopsy specimens. Mention of surgical removal or "resection" of bronchial cysts may be found in the paper by Walzel and in the case records of the Massachusetts General Hospital. Heuer and Andrus collected from the literature five cases in which a bronchial cyst was removed at the first operation and three others in which excision followed drainage or marsupialization.

Mixter and Clifford excised a gastric cyst after preliminary drainage, the patient being a male, aged 20 months, but they suggest that complete removal of the cyst may not have been accomplished. Nichols has reported the very interesting and instructive story of a girl who at the age of 4 years had a thoracotomy (right side) for "empyema." Subsequently, she was subjected to a series of deforming operations in the attempt to cure her "chronic empyema." When seen by Nichols at the age of eight years, it was finally determined that the "empyema" was in reality a cyst. The cyst was excised and the patient recovered. The most noteworthy event in the postoperative period occurred in the laboratory when the pathologist excitedly summoned the surgeon in the belief that part of the stomach had been removed. Nichols case is unquestionably one of the gastric type of cyst.

The present report includes an example of a gastric or gastrointestinal cyst which was completely extirpated after preliminary drainage.

SUMMARY

Congenital cysts of the mediastinum have been discussed. Special attention has been directed toward the bronchial and gastrointestinal cysts. These "entodermal" cysts differ from the teratomas in that they do not contain tissue derived from ectoderm; they reproduce the structures of the bronchus, stomach, or bowel; they occupy a posterior position in the mediastinum; they often cause symptoms, serious complications, or death in early infancy; and they are believed to originate from the foregut, from the respiratory buds or their derivatives (bronchial cysts), or from the omphalomesenteric duct (gastric and enteric cysts).

By contrast, the teratomas contain tissue derived from ectoderm (usually mesoderm, often entoderm); they have the structure that the name dermoid or teratoma implies; they arise in the anterior mediastinum; they frequently produce no symptoms for a period of 20 to 40 years; and they are believed to develop from cells which have become segregated early in embryonic life.

Case summaries of three mediastinal cysts have been reported, two of which were surgical specimens, one being a gastric cyst. Surgical principles have been outlined and exteriorization and drainage of cysts have been discussed.

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SELF-RETAINING SCAPULA RETRACTING FOR THORACOPLASTY WITHOUT USE OF A SPECIAL INSTRUMENT

O. C. Brantigan, M.D., and T. B. Aycock, M.D.
Baltimore, Md.

THE importance of adequate exposure in thoracoplasties and the difficulty in obtaining it is reflected by the number of scapular retractors which have been devised and which now are on the market.

The manual retractors designed for the scapula as described by Hudson,[3] and Mattison and Upshaw,[4] and those listed in current surgical instrument catalogues are all quite adequate and serve their purpose. It is doubtful if manual retracting will ever be completely superseded by mechanical retraction.

Self-retaining retractors have found a lasting place in various surgical fields, especially in abdominal operations. Such retracting is more important in thoracic surgery, particularly where retracting of the scapula is required.

Adequate exposure is of prime importance in all surgery, especially during the first stage of a modern thoracoplasty. It is of greatest importance in an operation such as an apicolysis. If the patient is strong and muscular, it is almost impossible for an assistant, regardless of how willing, to hold the scapula steadily, efficiently, and evenly. Each time the pull on the manual retractor is relaxed and then reapplied, trauma is being inflicted which might manifest itself in an increased evidence of shock, postoperative pain, or infection. Steady, efficient retraction will lessen trauma and allow the surgeon to work with greater speed, ease, and exactness.

The need for a self-retaining scapula retractor has been fulfilled by the ingenuity of Bethune,[1] Hall,[2] Overholt,[5] and Rø.[6] For countertraction, all of these men have utilized the operating table except Hall,[2] who made use of the body weight of the assistant. The efficiency of these is not denied but each is a special instrument and all have the same serious fault; namely, there is no fixed relationship between the part to be retracted and the source of countertraction. As stated by Overholt,[5] a shift in the position of the patient alters the retraction of the scapula.

The use of a rib as countertraction eliminates the need for a special instrument and it establishes a fixed relationship between the part retracted and the source of countertraction.

From the Department of Surgery, University of Maryland, and Mount Wilson Sanatorium.
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In performing an upper stage thoracoplasty the rib lower than the number proposed for removal is selected. The upper edge and inner surface is freed subperiosteally for a distance of approximately two inches, extending from the ileocostalis dorsi toward the front (Fig. 1). This allows countertraction for one blade of the retractor. The other blade is applied to the scapula covered by its muscles. The retractor must then be steadied and held in the desired position by the assistant (Fig. 2).

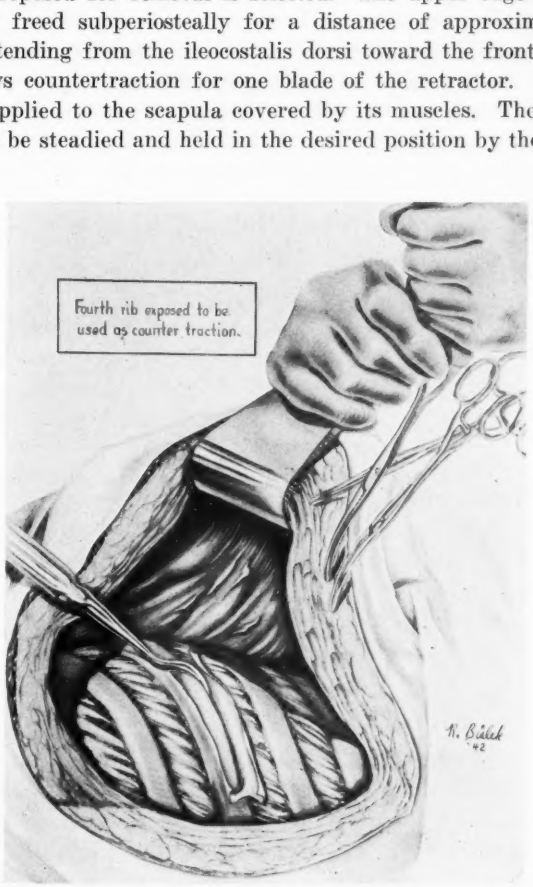

Fig. 1.—The drawing illustrates the method of preparing a rib for countertraction. When performing a first stage thoracoplasty of two or three ribs, the fourth rib is selected for countertraction. A lower rib may be used; in fact, if desired, the scapula may thus be retracted for a second stage thoracoplasty, using the eighth rib when resecting the fourth to the seventh ribs.

Self-retaining rib retractors or rib spreaders, usually found as standard equipment in any operating room used for thoracic surgery, can be efficiently utilized as a self-retaining scapula retractor. Two common types are illustrated in Fig. 2.

We have used this method of scapular retraction and found it quite satisfactory. Bethune's[1] words can be borrowed aptly, "The exposure it gives the first rib is a new experience to those surgeons who have been accustomed to manual retraction."

It is believed that the patients have experienced less postoperative pain and that wounds have healed better when mechanical scapular retraction was used.

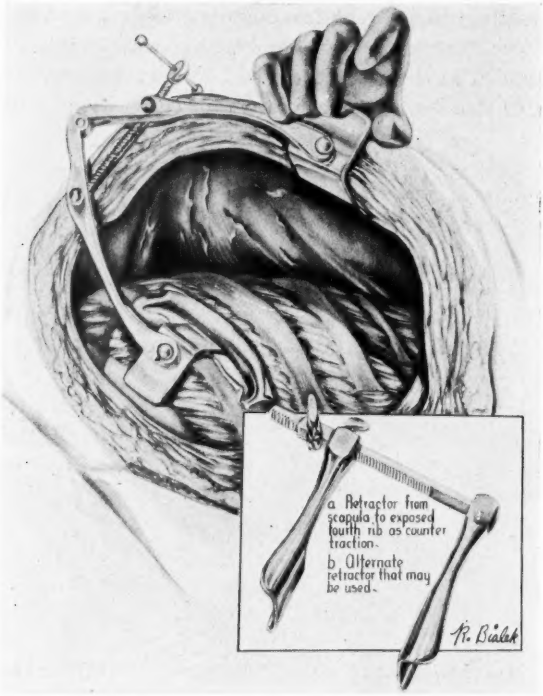

Fig. 2.—The drawing illustrates self-retaining scapula retracting by a standard rib spreader. The retractor blades extend from the exposed rib to the scapula. The small blade is used on the rib side, and the underlying lung is protected by a gauze sponge, not shown in the drawing. The large blade is used on the scapular side. If the retractor blade is serrated or fenestrated, it will not slip on the scapula.

CONCLUSION

When a rib is used as countertraction, the ordinary self-retaining rib spreader or retractor can be used as an efficient self-retaining scapular retractor.

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